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Table of Contents on Pages A-3 and A-4

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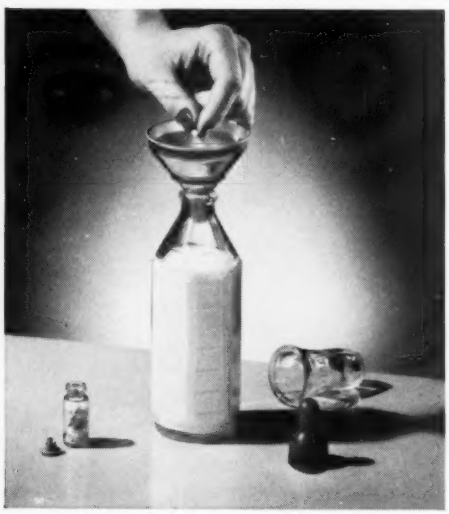

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Table *of* Contents—April 1951

VOL. IX, NO. IV

Special Articles

Coronary Occlusion and Coronary Insufficiency in 1000 Consecutive Cardiac Consultations.
ARTHUR M. MASTER, M.D. AND HARRY L. JAFFE, M.D. 279

Acute, Recurring and Chronic Pancreatitis.
JAMES T. PRIESTLEY, M.D. 287

Highlights in Medical Ophthalmology.
CONRAD BERENS, M.D. 293

The Pathology and Present Treatment of Ovarian Cysts.
RALPH C. BENSON, M.D. 313

Diagnostic Clinics

Treatment of High Diastolic Type of Essential Hypertension; Combined Use of Thiocyanates and Sympathectomy.
LOYAL DAVIS, M.D. 321

Diagnosis and Treatment of Polycythemia Vera.
ERNEST H. FALCONER, M.D. 325

The Significance of Frequency of Urination.
HERMAN L. KRETSCHMER, M.D. 331

Oral Health in Children.
JOHN A. BIGLER, M.D. 333

Case Report

Complications and Surgical Indications in Chronic Ulcerative Colitis.
MAURICE A. MICHAEL, M.D. 340

General Practice Review

Encephalitis. SHERMAN F. GILPIN, JR., M.D. 345

Treatment of Contact Dermatitis. JOHN GODWIN DOWNING, M.D. 347

Treatment of Acute Cholecystitis. WARREN H. COLE, M.D. 349

(Continued on page A-6)

Table of Contents — *Continued*

Pediatrics in General Practice

Treatment of Mumps. EDWARD B. SHAW, M.D. 353
Treatment of Bronchiectasis. GEORGE L. WALDBOTT, M.D. 356
Hemorrhagic Disease of the Newborn. A. H. PARMELEE, M.D. 358

Scientific Exhibit

Technic of Tracheotomy.
A. C. STUTSMAN, M.D. 360

Departments

EDITORIALS 364

The Scientific Method in Medical Research
Foundations, Corsets and Brassieres
Academic Humor

MEN OF MEDICINE. *Arthur Krida, M.D.* 368

LEAVES FROM A DOCTOR'S DIARY. Maurice Chideckel, M.D. A-10

PHOTOGRAPHIC SALON PRIZEWINNERS A-18

CONSULTATION SERVICE A-24

Effect of Antibiotics on Immunity Against Pertussis
Retrolental Fibroplasia in Premature Births
Suppression of Lactation and Breast Pathology
Post-Zoster Pain

NEW DRUGS AND INSTRUMENTS A-51

DR. PEPYS' PAGES. Morris Fishbein, M.D. A-56

THE MEDICAL BOOKMAN A-82

CORRESPONDENCE DEPARTMENT A-92

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Coronary Occlusion and Coronary Insufficiency

ARTHUR M. MASTER* AND HARRY L. JAFFE

COLUMBIA UNIVERSITY COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK

Acute coronary attacks may be divided clinically, electrocardiographically, pathologically and therapeutically into (1) classical angina pectoris, (2) acute coronary insufficiency and (3) acute coronary occlusion. In 1000 consecutive cardiac consultations, 450 of 618 cases with organic heart disease had coronary artery disease. Of these 450 cases, 38 per cent had chronic coronary insufficiency (angina pectoris or congestive failure), 26 per cent had acute coronary insufficiency, and 36 per cent had acute coronary occlusion. The differentiation between coronary occlusion and insufficiency which can be made electrocardiographically in 95 per cent of cases is discussed in this paper.

PRECORDIAL pain or angina caused by coronary artery disease has occupied the attention of physicians for several centuries and today is probably the most important single symptom in medicine. It has been estimated that as many as 8,000,000 persons in this country have heart disease[1] and in more than half, it is coronary in origin. As the average span of life continues to increase, the incidence of coronary heart disease will also rise. We have shown that about 1,000,000 attacks of coronary occlusion occur annually[2] and it is probable that an equal number of other acute coronary episodes, which we call instances of coronary insufficiency, also take place.

It is only during the past 35 years that it has been possible to correlate the various types of acute coronary episodes with pathologic and physiologic observations.

Until the past decade acute chest pain assumed to be due to involvement of the coronary arteries was diagnosed either as angina pectoris or as coronary occlusion. If the attack directly followed physical effort or emotional strain, lasted less than 15 or 30 minutes, and was relieved by nitroglycerin, and if the recovery was complete, it was called angina pectoris and was assumed to indicate a temporary, functional insufficiency of the coronary flow. An RS-T depression and/or T-wave alteration sometimes appeared in the electrocardiogram during the attack and promptly disappeared.

If the pain lasted longer than an hour, began at rest, was not relieved by nitroglycerin, and was accompanied by vomiting, shock, drop in blood pressure, pericardial rub, gallop rhythm or heart failure, the attack was classified as coronary occlusion with myocardial infarction. This diagnosis was confirmed by the characteristic electrocardiographic pattern of RS-T elevation and Q-waves with serial change into T-wave inversion. Fever, leukocytosis and abnormal sedimentation rate were also present.

A typical instance of coronary occlusion was the case of J. S., a 58-year-old executive who had experienced a coronary occlusion with anterior infarction in 1948. He consulted us for the first time on September 28, 1949, because of angina following effort. At that time his blood pressure was 150/96 and his electrocardiogram showed the residual changes of a coronary occlusion

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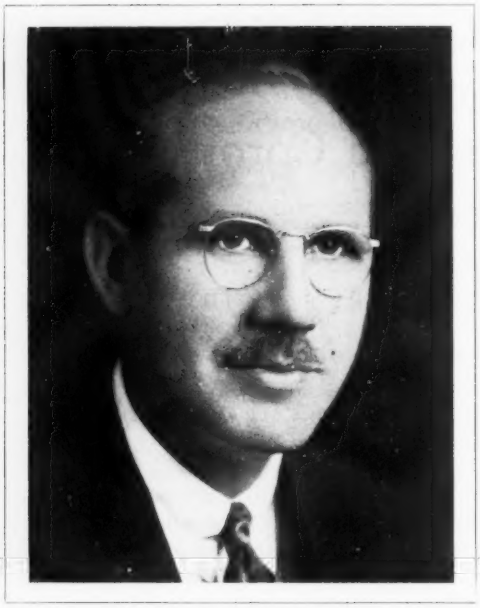

ARTHUR M. MASTER

HARRY L. JAFFE

with anterior infarction. On December 21, 1949, he complained of severe abdominal cramps, diarrhea, and pain in the left shoulder.

Several hours later he developed substernal and epigastric pain and sweating. A gallop rhythm and pericardial rub were heard. His blood pressure fell to 110/60. His electrocardiogram now showed the characteristic changes of a recent coronary occlusion with posterolateral infarction, i.e., deep Q-waves and RS-T elevation in leads II, III and aVF and RS-T depression in leads V2-4, and RS-T elevation in leads V5, 6 (Figure 1). On the second day his temperature was 101° F. and his white blood count 16,000. On the third day his sedimentation rate was 62 mm. He made an uneventful recovery and was permitted to leave his bed after three weeks. At this time his electrocardiogram showed a typical Q_3-T_3 pattern and his sedimentation rate was still elevated to 32 mm.

There were some cases of coronary occlusion, which were diagnosed electrocardiographically, that did not evidence the typical clinical signs. Not infrequently the pain was mild and of short duration, as in simple angina pectoris. Occasionally the occlusion was asymptomatic and its occurrence was unsuspected until later when a routine electrocardiogram was taken. The explanation for occlusion without symptoms is based on the assumption of a previously well developed anastomotic circulation.

In recent years it has become evident that many acute coronary episodes cannot be classified either as angina pectoris or as coronary occlusion. In these cases the pain is more severe and prolonged than in angina pectoris; there may be a drop in blood pressure, fever, leukocytosis and an abnormal sedimentation rate, without the electrocardiographic pattern of coronary occlusion. Instead RS-T depression and/or T-wave inversion may appear. There is no pericardial rub or peripheral embolization.[3-8]

A typical instance was the case of N. G., a man of 53, who had a history of duodenal ulcer for many years. While lifting crates of vegetables weighing 30 to 50 pounds each, he suddenly developed severe substernal pain and profuse perspiration. Neither gallop rhythm nor pericardial rub was present. The electrocardiogram showed RS-T depression and T-wave inversion in leads I and V_4. There were no Q-waves or RS-T elevations (Figure 2). The next day his temperature was 100.6° F. and on the third day his sedimentation rate was 25 mm.

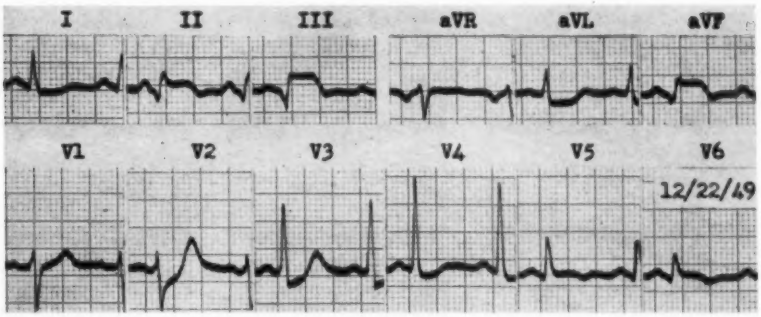

FIGURE 1. J. S., m., 58 (15283-M). Acute coronary occlusion with posterolateral infarction.

There was no leukocytosis. His illness followed a benign course but his electrocardiogram readings gradually returned to normal in six months. In this case subendocardial necrosis was undoubtedly present.

Another instance of acute coronary insufficiency is the case of H. A. K., a man of 54. Following very strenuous physical exertion for several days, he experienced severe substernal pain and went into mild collapse. The electrocardiogram revealed RS-T depression and diphasic T-waves in the standard leads (Figure 3). He quickly recovered and the next day his electrocardiogram was normal. There was no fever, leukocytosis or alteration in the sedimentation rate. In this case there was acute myocardial ischemia but probably no necrosis.

When death occurs in these cases no acute changes are found in the coronary arteries but focal areas of necrosis may be present in the subendocardial layer and papillary muscles. We employ the term "acute coronary insufficiency" for this group of cases. They represent a more advanced degree of coronary insufficiency than the classical anginal attack. In the severe cases, in which subendocardial necrosis occurs, the electrocardiographic changes may persist for several weeks. In less severe cases, if electrocardiographic alterations occur, they usually disappear within several days. If they remain longer than five or seven days necrosis has probably taken place.

The vast majority of persons who have acute coronary insufficiency recover from the attack but in others the disease pursues a severe course and death may occur. In fact, it has been conclusively shown that sudden deaths from coronary disease usually result from acute coronary insufficiency without occlusion.[9,10]

During the first few days of an acute coronary episode it may be difficult for the physician to distinguish between acute coronary insufficiency and the premonitory phase of coronary occlusion.[11] Almost half the patients with coronary occlusion experience premonitory pain for days or weeks during which period the electrocardiographic pattern of coronary insufficiency, that is, RS-T depression and T-wave inversion, may be present. At this time, it is impossible to foretell whether the condition will subside or lead to occlusion with through and through infarction.

One clue to the differentiation between coronary insufficiency and coronary occlusion is the mode of onset. Coronary occlusion occurs at rest or during routine activity in 98 per cent of cases.[12,13] It is now generally accepted that the attack is fortuitous and spontaneous. It is our opinion that in the occasional case in which the attack directly follows effort or trauma the association is coincidental. Coronary insufficiency also occurs spontaneously in more than half the cases but it is often precipitated by factors producing sudden ischemia of the myocardium.[6-8] There are three mechanisms by which this may occur: (1) increased demand of the myocardium for oxygen, (2) reduced coronary flow, and (3) diminished oxygen-carrying capacity of the blood. In most instances two or all three conditions combine to produce insufficiency. The circumstances attending the onset of 100 attacks of coronary insufficiency recently observed by us are shown in Table 1.

When an acute coronary episode follows a competent precipitating factor the diagnosis is almost always coronary insufficiency. Only in rare instances is a specific precipitating factor coincidental with coronary occlusion. This opin-

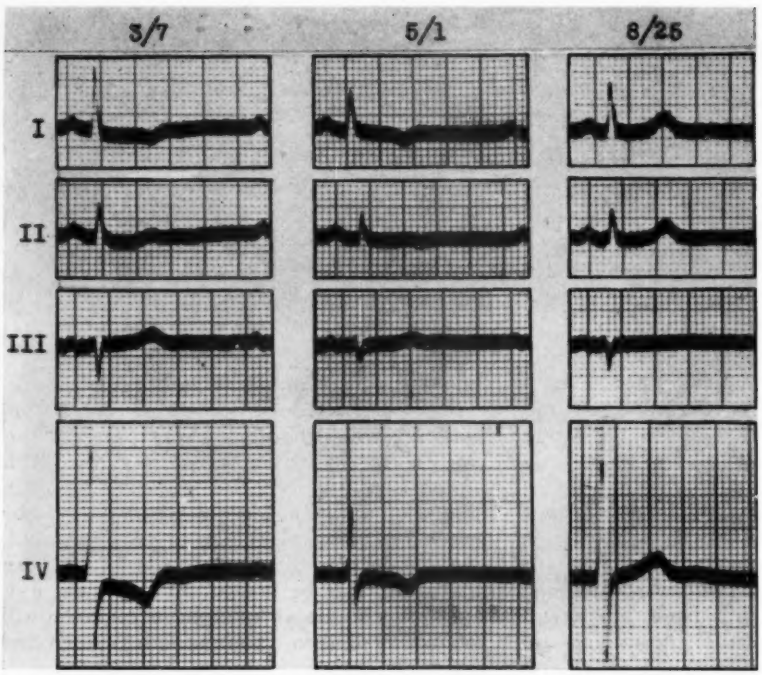

FIGURE 2. N. G., m., 53. Acute coronary insufficiency following physical exertion.

TABLE 1

ACTIVITY AT ONSET OF CORONARY INSUFFICIENCY
100 ATTACKS

Spontaneous	56	Acute cholecystitis	1
Hemorrhage	10	Acute pancreatitis	1
Pulmonary embolism	8	Gastroenteritis	1
Unusual effort	6	Hypertensive crisis	1
Tachycardia	3	Postsympathectomy	1
Surgical procedure	3	Carbon monoxide	1
Excitement	3	Tobacco and alcohol	1
Meal	2	Addisonian crisis	1
Trauma	1	Asthma	1
Acute left heart failure	1		

ion was recently corroborated by Clark[10] who examined the heart in 15 cases in which death occurred suddenly during severe exertion. He found coronary sclerosis in each case but no instance of acute occlusion. When an attack occurs spontaneously it is usually coronary occlusion, but it may be coronary insufficiency.

The high incidence of acute coronary insufficiency has not generally been recognized. Many attacks are too mild to require medical care or are diagnosed as angina pectoris or coronary occlusion. For example, Mrs. R. S., a housewife of 56 with a history of diabetes and occasional angina on effort, experienced moderately severe precordial pain. It lasted 45 minutes until her physician administered a hypodermic of DEMEROL. The blood pressure, which usually varied between 130 and 150 systolic, was 200/110. She slept all night and in the morning felt well. A 12 lead electrocardiogram proved to be normal and remained so two days later. At this time her blood pressure was 190/100 and her temperature was normal. It was concluded that her illness had been an anginal episode and she was permitted out of bed. However, the next day the diagnosis had to be changed to acute coronary insufficiency when the sedimentation rate was reported 49 mm., indicating subendocardial necrosis or infarction. This was confirmed electrocardiographically on the eighth day when the T-wave became diphasic in V5, 6. The T-wave returned to normal in two weeks.

It has been our clinical impression that acute coronary insufficiency, including both spontaneous and precipitated cases, is at least as common as coronary occlusion. Therefore, we reviewed the diagnoses in 1000 consecutive private patients seen in cardiac consultations. The period covered was 1946-1948.

From Table 2 it will be seen that 618 persons, or almost two-thirds, were found to have or-

TABLE 2

PRIMARY DIAGNOSES IN 618 CASES OF ORGANIC CARDIAC DISEASES

		Percentage	CASES	
			Number	*Per Cent*
Coronary disease			450	73
Chronic (angina pectoris)	171	38		
Acute coronary insufficiency	115	26		
Acute coronary occlusion	164	36		
Hypertension, severe*			78	13
Chronic cardiovalvular disease†			63	10
Congenital heart disease			16	3
Miscellaneous			11	2

*Hypertension was present in 205 or 33 per cent.
†Chronic cardiovalvular disease was present in 80 or 13 per cent.

ganic heart disease and that one-third had functional diseases. These figures emphasize the high incidence of functional heart disturbances and conditions simulating cardiac disease.

Almost three quarters of the organic cases had coronary artery disease. Thirty-eight per cent of these had anginal syndrome or heart failure, i.e., chronic coronary insufficiency. Twenty-six per cent had experienced at least one episode of acute coronary insufficiency and many of these also had an anginal syndrome. The remaining 36 per cent had developed one or more coronary occlusions. These figures would seem at first to indicate that the incidence of coronary occlusion is slightly higher than that of coronary insufficiency. Actually, they suggest that coronary insufficiency is more frequent since it is probable, as we have stated, that a considerable number of coronary insufficiency episodes are not severe enough to prompt the victim to seek medical attention or are improperly diagnosed. This is true particularly of precipitated cases in which the causative factor may mask the signs and symptoms of coronary insufficiency. For example, hemorrhage, postoperative shock, tachycardia and heart failure usually overshadow the associated coronary inadequacy and myocardial ischemia. To cite a case, T. L., a girl of 19 with severe ulcerative colitis, bled profusely from the rectum and her hemoglobin fell to 3 gm. She deteriorated rapidly and died. At necropsy necrosis of the papillary muscles was found but the coronary arteries were discovered to be normal.[14]

Some physicians employ the term myocardial infarction, avoiding the nomenclature of coronary occlusion and insufficiency on the assumption that the two cannot be differentiated clinically.[15] Although this is true in exceptional cases, differentiation can be made electrocardiographically in 95 per cent.[6-8] Postmortem studies have shown that if an attack is associated with Q-waves and RS-T elevation, coronary occlusion is almost always present and the infarction is confluent and large. On the other hand, if the electrocardiogram shows RS-T depression or T-wave inversion, no occlusion is present and there may be scattered areas of necrosis in the subendocardial region and papillary muscles.

THE exceptions to these rules include (1) coronary occlusion with true posterior or lateral infarction (as distinguished from the common diaphragmatic infarction) which shows only RS-T depression and T-wave inversion. The correct diagnosis of coronary occlusion with posterior infarction is suggested by the presence of a high R-wave in the right precordial leads, i.e., $V_{1, 2}$. This tall R should be sought in electrocardiograms otherwise suggestive of coronary insufficiency. (2) Coronary insufficiency of such severe degree as to cause ischemia or infarction of the entire myocardium and therefore RS-T elevation and Q-waves. For example, H. K., a man of 46, suddenly went into shock 17 days after cholecystectomy and his electrocardiogram showed a large Q-wave and RS-T elevation in lead III. Death occurred after 10 hours and necropsy showed no fresh change in the myocardium or coronary arteries. The latter showed only moderate narrowing. (3) A transitory RS-T elevation, instead of the usual RS-T depression, in cases of coronary insufficiency. This may be caused by exercise, particularly in patients who have sustained a previous coronary occlusion. The elevation promptly disappears.

These exceptional cases are certainly less than 5 per cent of the total. Ordinarily, in acute coronary insufficiency no deep Q-waves are present and RS-T elevations are absent.

A disadvantage of discarding the terms coronary occlusion and insufficiency in favor of myocardial infarction is that this nomenclature does not enable one to distinguish between two different types of infarction. Nor does it enable one to recognize the severe coronary episodes without infarction or occlusion, particular-

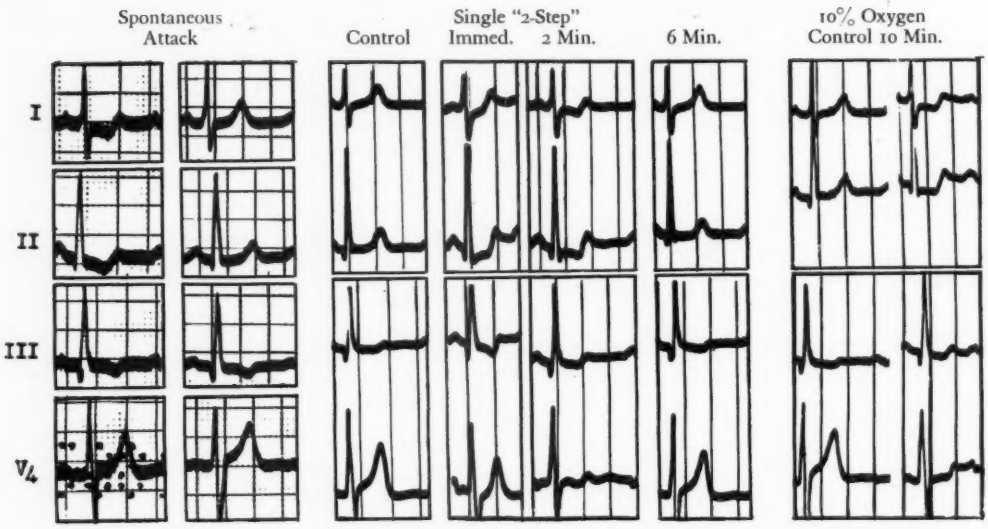

FIGURE 3. H. K., m., 54. Acute coronary insufficiency following physical exertion. RS-T depression in leads I and II and T-wave inversion in leads I, II and III. Return to normal in one day. At a later date, almost identical changes were induced by the single "two-step" exercise test and the 10 per cent oxygen test.

ly those induced by factors causing myocardial ischemia. We believe the term acute coronary insufficiency is useful if it is employed to connote a coronary attack without occlusion, more severe than a simple, transitory anginal episode, and usually associated with RS-T depression and/or T-wave inversion for several days or weeks, depending upon the absence or presence of myocardial necrosis.

In our opinion, it is valuable to differentiate between coronary occlusion and insufficiency for purposes of diagnosis, prognosis and, particularly, therapy.[16]

Diagnostically, it is important to recognize that many extracardiac conditions, for example, acute cholecystitis or other abdominal conditions, may cause reflex coronary insufficiency with cardiac symptoms and acute changes of coronary insufficiency in the electrocardiogram. The presence of these changes should not militate against correct diagnosis of cholecystitis, etc.

The prognosis for the immediate attack is usually excellent in the spontaneous type of coronary insufficiency. Indeed, postmortem material is difficult to collect. However, death may occur and we have already alluded to sudden deaths from this type of attack. In coronary insufficiency precipitated by effort, emotion, hemorrhage, shock, etc., the prognosis depends upon the severity of the etiologic condition and the promptness and adequacy of treatment.

This type of coronary insufficiency is a therapeutic, clinical entity. Even more important, it is essential to try to prevent its development. Therefore, persons with coronary and other types of heart disease should be cautioned to avoid situations which suddenly increase the work of the heart: for example, unusual exertion, emotional disturbance, trauma, straining at stool, and excessive eating, drinking, or smoking, particularly in combination. Extremes of cold or heat are dangerous to heart disease patients. If possible they should move to more temperate regions during the winter or summer if cold or heat causes significant symptoms. It may be necessary for them to curtail sexual intercourse. Hyperthyroidism should be treated with iodine.[131]

Another cause of coronary insufficiency is diminished coronary flow. Precaution should be taken to prevent this in operative procedures. Careful selection and administration of anesthesia in combination with oxygen and intravenous fluids and transfusions usually prevent postoperative shock and hemorrhage and enable even severe cardiac patients to undergo the most serious operations successfully. When spinal anesthesia is employed, NEOSYNEPHRINE may

prevent a drop in blood pressure and, therefore, coronary insufficiency. Paroxysmal tachycardias may be avoided during operation by administering PRONESTYL or quinidine before or during the operation. In cardiac patients large doses of insulin should be avoided to prevent hypoglycemia.

Should coronary insufficiency appear in any of these conditions, prompt and energetic treatment of the precipitating factor, such as shock or tachycardia, is required and may be lifesaving. This holds for other conditions reducing the coronary blood flow, such as heart failure, infections including gastroenteritis, Addison's disease and acute abdominal conditions, e.g., cholecystitis. Early ambulation and anticoagulants may prevent pulmonary embolism.

Coronary insufficiency may also result from a reduction in the oxygen-carrying capacity of the blood. This is true particularly of acute hemorrhage in any part of the body. In this type of coronary insufficiency, transfusions must be given promptly and repeated as often as necessary. We have given cardiac patients as much as 2500 cc. of blood within 24 hours during and after prostatectomy or in DICUMAROL bleeding. Pulmonary edema sets in on rare occasion but this can usually be overcome by an intravenous mercurial diuretic. This consideration must not militate against the administration of blood in large amounts if it is required. High altitudes may also interfere with adequate oxygenation. Air travel is usually very well tolerated by cardiac patients but it is not advisable for them to live permanently or exert themselves at altitudes above 7000 feet. In carbon monoxide poisoning oxygen is very useful.

Neither coronary occlusion nor the spontaneous type of coronary insufficiency can be prevented, and treatment is essentially symptomatic, i.e., bed rest, relief of pain, and a low calorie diet, unless a complication arises. Congestive failure, arrhythmias, and pulmonary and peripheral emboli, for example, are treated when they occur. We employ penicillin and anticoagulants routinely in coronary occlusion. We also frequently administer anticoagulants in spontaneous coronary insufficiency when bed rest for more than 10 days is indicated, to prevent pulmonary infarction. Another possible reason for giving anticoagulants in coronary insufficiency is to avoid completion of any occlusion that may be forming. However, it will be difficult to evaluate the results of this practice since coronary insufficiency patients usually recover spontaneously. To determine the efficiency of anticoagulants, very large series of cases will have to be observed.

ILLUSTRATIVE CASES OF CORONARY INSUFFICIENCY

In the first 2 cases acute coronary insufficiency developed spontaneously.

Case 1—I. F., a very neurotic storekeeper of 46, suddenly experienced severe substernal pain following his usual evening meal. The pain lasted more than an hour. His physician gave him a hypodermic and he slept all night. In the morning he was free of pain and felt quite well. Physical examination was negative and his blood pressure was, as usual, about 120/80. His temperature was normal.

The electrocardiogram showed slightly inverted T-waves in all the limb and chest leads. Two days later the T-waves were more deeply inverted but were approximately normal by the seventh day. The patient was permitted out of bed. After going to the bathroom, he complained of precordial pain and the electrocardiogram again showed T-wave inversion. These persisted for two weeks, at which time the electrocardiogram became normal. He has remained well for two years.

Case 2—P. T., an obese salesman of 61, suddenly experienced some substernal pain while at home at 10:00 P.M. He had been driven into the country for the day, had returned about 6:00 P.M. and had a moderate-sized dinner. The electrocardiogram showed slight T-wave inversion in leads I and $V_{2,4,5}$. His temperature was 100.6° F. and his sedimentation rate 28 mm. The disease ran a very uneventful course. The T-waves gradually became moderately inverted. The patient was in bed three weeks and the electrocardiogram returned to normal in two months. He was asymptomatic during the following seven months and then developed an acute coronary occlusion with anterior infarction.

In the following case, coronary insufficiency was precipitated by hyperthyroidism.

Case 3—Mrs. S. S., aged 62, complained of increasingly severe angina of several months' duration. She then developed status anginosus which did not respond to nitroglycerin or large doses of opiates. The electrocardiogram showed varying T-wave changes. At first she was considered to have suffered a major infarction but her temperature, blood count and sedimentation rate were normal. Hyperthyroidism was then suspected and this was confirmed when she excreted only 15 per cent of a test dose of I^{131}. She was then given a therapeutic dose of this substance and, since its effect is not excreted for four to six weeks, Lugol's solution was also administered. Within 10 days her pain began to subside and the electrocardiogram became stabilized.

The next case demonstrated the occurrence of

coronary insufficiency following sudden hemorrhage.

Case 4—A. Z., a storekeeper of 62, suddenly felt dizzy and later passed a tarry stool. In two days his hemoglobin dropped 14 gm. to 8 gm. He did not complain of chest discomfort but the electrocardiogram showed slight RS-T depression and T-wave inversion in leads I, II, and V_4, 5 and 6. His temperature was 100° F. and his sedimentation rate was normal. The electrocardiographic changes increased moderately for several days and returned to normal in two weeks.

In the following case coronary insufficiency developed after acute enteritis. This is not uncommon.

Case 5—C. N., a man of 65, developed a severe diarrhea lasting several days. For the first time, he complained of precordial pain. The electrocardiogram showed RS-T depression and T-wave inversion in several leads. Within one week the readings returned to normal.

The next case illustrates the occurrence of transitory RS-T elevation instead of depression in coronary insufficiency. It occurs in two or three per cent of cases. In this case the coronary insufficiency was induced by tachycardia.

Case 6—L. P., a storekeeper of 58, had developed a coronary occlusion and posterior infarction two years previously. He entered the hospital because of precordial distress and palpitation. Examination revealed auricular fibrillation with a rapid ventricular rate. Electrocardiograms showed RS-T elevation in lead III which had not been present in his previous record. There was no fever or abnormal sedimentation rate. The auricular fibrillation disappeared spontaneously within several hours and the ventricular rate dropped to 80. At this time, the RS-T elevation in lead III was no longer present, indicating that it had been caused by coronary insufficiency.

In the following case coronary insufficiency developed after operation. This is a common occurrence. It should be remembered that occasionally T-wave changes occur after operation without any corresponding clinical signs. In such cases, the electrocardiographic changes are probably related to electrolyte changes.

Case 7—F. C., an executive, 65 years old, underwent prostatectomy in one stage. General ether anesthesia was used. Shortly after returning to his room by stretcher, he was found to be in a state of mild collapse. His electrocardiogram showed marked T-wave inversion in leads I and V_4. With supportive treatment, his condition rapidly improved and the electrocardiogram returned to normal in five days.

In the next case, emotion and exertion precipitated coronary insufficiency.

Case 8—G. K., a lawyer of 44, was suddenly called to see his mother who was seriously ill several hundred miles away. He drove for eight hours and then found that she had died. That evening he developed severe precordial pain. His electrocardiogram showed deeply inverted T-waves in leads I, II and $V_{2, 4,}$ and 5. In two days the electrocardiogram had returned to normal. There was no change in temperature, blood count or sedimentation rate.

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Acute, Recurring and Chronic Pancreatitis

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MAYO CLINIC, ROCHESTER

Pancreatitis is receiving an increasing amount of attention from the internist and surgeon alike. There are three main types: (1) acute pancreatitis, (2) recurring or relapsing pancreatitis and (3) chronic pancreatitis. The clinical and surgical features of these three forms of the disease are considered in this paper.

Although much has been learned concerning the recognition of pancreatic disease during the last few decades, much still remains to be learned of the pathologic physiology and treatment of various diseases of this gland. At the present time pancreatitis is a condition which is receiving an increasing amount of attention from the internist and surgeon alike. Before any of the details of this condition are discussed, it might be well to classify the various types of pancreatitis which are encountered. In brief, there are three main types: (1) acute pancreatitis, (2) recurring or relapsing pancreatitis and (3) chronic pancreatitis.

ACUTE PANCREATITIS

Acute pancreatitis is a condition that has been recognized for many years. It is commonly subdivided into the more severe form known as acute hemorrhagic pancreatitis or acute pancreatic necrosis on the one hand and acute interstitial pancreatitis or acute pancreatic edema on the other hand. Probably both of these types of pancreatitis are similar in etiology and general characteristics, the former being more severe than the latter. Depending upon the severity of the disease, abdominal pain, distention, rigidity, nausea, vomiting and shock may be more or less pronounced.

Although in past years it was common practice to advise surgical treatment for acute pancreatitis, it is now generally conceded that medical or conservative management is preferable. General supportive measures consisting of rest in bed, sedation and adequate administration of parenteral fluids and antibiotics are commonly employed. Although antibiotics are always used, their actual value appears to be somewhat questionable as it has not been definitely determined that this disease is of infectious origin. Inasmuch as opinion is relatively uniform regarding the treatment of this condition, the present remarks will be confined for the most part to discussion of the other types of pancreatitis about which there is less uniformity of opinion.

RELAPSING OR RECURRING PANCREATITIS

Relapsing or recurring pancreatitis is a definite clinical entity and a serious disease which is being recognized with increasing frequency. It is characterized by recurring attacks of acute

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or subacute pancreatitis of greater or lesser severity and greater or lesser frequency. It may lead to virtual destruction of the parenchyma of the pancreas as well as to various other complications. While there seems to be a definite relationship between recurring pancreatitis and disease in the biliary tract, the exact relationship between these two conditions is not clear. Gambill and his associates[1] have reported that approximately one half of the patients who have recurring pancreatitis have associated disease in the biliary tract. On the other hand there are certain definite differences between patients who have recurring pancreatitis and those who have calculous disease in the biliary tract. For example, 4 out of 5 patients with recurring pancreatitis are men.

Although recurring pancreatitis may occur at any age, it is more common during the third and fourth decades of life. Obese patients are not particularly susceptible. In these regards, obviously, those patients who have recurring pancreatitis differ somewhat from those who have calculous disease in the biliary tract. If the two conditions exist simultaneously, the patient generally presents symptoms of each disease, one being superimposed upon the other.

Acute attacks—The acute attacks of relapsing pancreatitis are characterized by severe pain in the upper part of the abdomen and located in descending order of frequency in the epigastrium, the right upper quadrant and the left upper quadrant of the abdomen. The pain is most suggestive of pancreatitis when it is located in the left upper quadrant of the abdomen, as relatively few other conditions cause pain in this location. Commonly pain is referred into the back at a level which corresponds with its location anteriorly. Most often this is at the level of the thoracolumbar region. While the pain is not abrupt in onset it develops with some rapidity to a high level of severity which continues as a constant pain until the attack gradually subsides.

A single hypodermic injection of morphine will seldom relieve the pain of pancreatitis in contrast with a usual attack of biliary colic. In fact, multiple hypodermic injections are generally required and even these may fail to keep the patient comfortable. Ordinarily the pain lasts much longer than the ordinary attack of biliary colic, as the majority of patients experience pain for at least 10 hours and the pain may persist for days or even several weeks. Excessive use of alcohol or a large meal frequently precipitates an attack. Most patients with recurring pancreatitis use a large amount of alcohol. Abdominal tenderness may persist for some days following an acute attack. Fever, nausea, vomiting and abdominal distention are commonly present.

From the point of view of laboratory diagnosis, determinations of the value for amylase and lipase in the serum are of value. Unfortunately, values for these enzymes remain elevated only a relatively short period of time following the onset of acute attacks, perhaps for one or two days. It should be realized that elevation of lipase and amylase in the serum is not pathognomonic of acute pancreatitis, as these values may be elevated in the presence of acute rupture of a duodenal ulcer, certain diseases of the liver and even after a hypodermic injection of morphine or codeine in some cases. Keith, Zollinger and McCleery[2] have reported that there is commonly an elevation of the value for amylase in peritoneal fluid in the presence of acute pancreatitis, which may persist for a week or even longer after the onset of an acute attack.

Stages of disease—In general, two main stages can be recognized in chronic recurring pancreatitis; namely, the early and the late stages. It is true that combinations of symptoms and findings may exist which make it difficult or impossible to determine whether a given patient has an early or a late stage of the disease, but usually this can be determined without too much difficulty.

The early stage of the disease is characterized by recurring attacks of acute pancreatitis with freedom from pain between the attacks. At this time there is seldom any evidence of advanced chronic changes in the pancreas which result in chronic pancreatic insufficiency.

In contrast, the late stage of the disease is characterized by virtually constant pain in the upper part of the abdomen associated with acute exacerbations. Narcotic addiction is common under these circumstances because of the severity and constancy of the pain. Generally the previous recurring acute attacks have resulted in advanced chronic changes in the gland which frequently culminate in virtually complete physiologic destruction of the pancreas with insufficiency of both internal and external secretions. Calcification may or may not be present in the pancreas.

FIGURE 1. Diffuse pancreatic calcification.

It is of some importance to determine which stage of the disease the patient has, as in the opinion of my colleagues and myself indications for treatment vary depending upon the stage of the disease that is present. In the early stage of recurring pancreatitis, treatment is directed toward preventing the recurring acute attacks and thereby preventing progressive damage to the pancreas which may result in pancreatic insufficiency. In the late stages of the disease, the main indication for treatment is relief of the patient's pain, as the pancreas has been largely destroyed and not much can be accomplished toward the preservation of pancreatic function.

Surgical treatment—Treatment of recurring pancreatitis is based upon the presumed cause of the disease. Although some doubt still exists concerning the cause of recurring pancreatitis, it is commonly thought that repeated reflux of bile from the lower end of the common bile duct into the pancreatic duct is responsible for this disease. Therefore, most forms of treatment for this condition, which are currently employed, are directed toward preventing this reflux.

The treatment of recurring pancreatitis may be most gratifying or most disappointing. The fact that so many forms of treatment have been used for this condition is in itself evidence that no single treatment has proved to be uniformly satisfactory. Some years ago, cholecystostomy and cholecystectomy were frequently used; however, it is now realized that these measures are ineffective. More recently, vagotomy and gastric resection have been suggested as methods of treating recurring pancreatitis on the basis that both of these procedures might result in a lessened stimulation of external pancreatic secretion. Neither of these procedures has proved to give satisfactory results in a significant group of cases over long periods of time. They are not recommended at this time.

For the most part, surgical treatment of recurring pancreatitis currently is directed toward preventing an increase in the intraluminal pressure within the common duct which might result in reflux of bile up the pancreatic duct. It has been our practice in most cases either to establish a prolonged external drainage of bile by inserting a T tube into the common bile duct and permitting it to remain for a period of approximately a year, or to establish an internal drainage of bile through an anastomosis between the biliary and intestinal tracts. Usually, external drainage of bile has been employed in the relatively mild case in which there is a history of rather infrequent attacks over a short

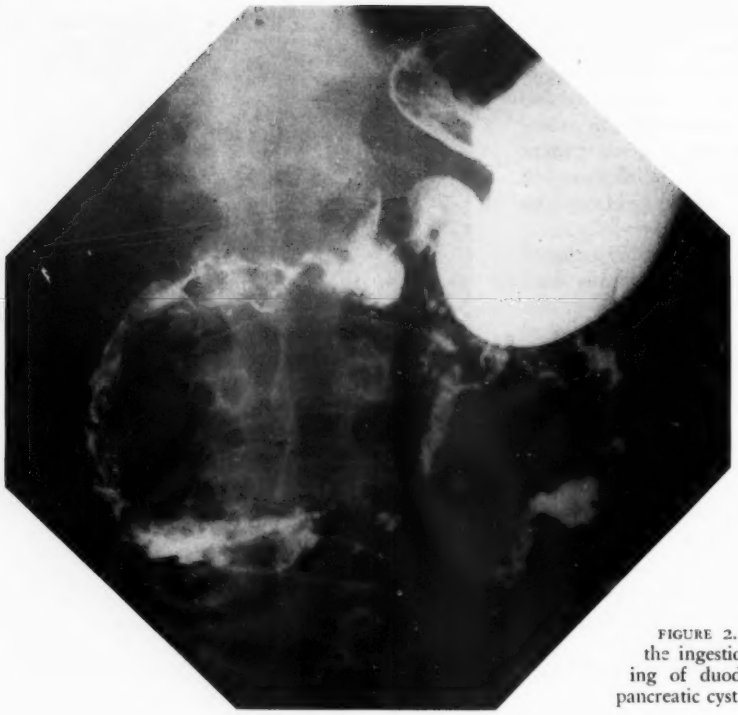

FIGURE 2. Roentgenogram, made after the ingestion of barium, shows "widening of duodenal loop," suggestive of a pancreatic cyst.

period of time and in which previous surgical procedures have not been employed. In contrast, internal drainage of bile such as by choledochoduodenostomy has been employed in the more advanced case in which attacks are frequent and in which more conservative measures have failed to give relief. Occasionally external drainage of bile affords relief of the patient's symptoms while the T tube is in place, but symptoms recur after removal of the T tube. Under these circumstances, choledochoduodenostomy or some similar type of procedure may give more lasting relief.

Doubilet and Mulholland[3] have reported a group of cases of recurring pancreatitis in which internal sphincterotomy was employed with early results that appear promising. In this procedure the sphincter of Oddi is incised by an instrument that is passed down the common duct. I have had no experience with this operation myself, but on several occasions recently I have performed a transduodenal incision of the sphincter of Oddi. This procedure has not been used in a large enough group of cases for an adequate period of time to permit its evaluation.

CHRONIC PANCREATITIS

Two main forms of chronic pancreatitis may be recognized. One is a mild form of the disease characterized by slight or moderate induration in the head of the pancreas which is noted at the time of cholecystectomy or some other abdominal operation. If this induration in the head of the pancreas is unassociated with other changes it is likely that it is not of great clinical significance. Generally, treatment is not indicated. However, if the common duct has been explored, the T tube may be left in place for a longer time than usual.

The second form of chronic pancreatitis is a very serious disease and is characterized by pronounced induration, usually present throughout the entire pancreas. This is caused by recurring attacks of acute or subacute pancreatitis and may result in virtually complete destruction of the pancreatic parenchyma. Under these circumstances, diabetes and external pancreatic insufficiency may exist. There may or may not be roentgenologic evidence of calcification in the pancreas (Figure 1). If present, this may be diffuse throughout the entire gland or localized

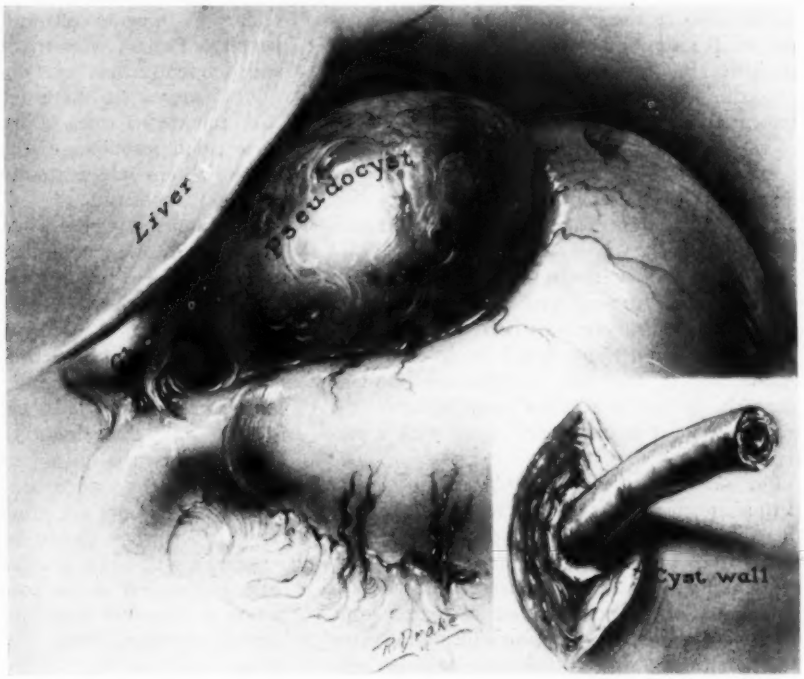

FIGURE 3. Drawing of a pseudocyst of the pancreas which presented above the lesser curvature of the stomach. The inset shows marsupialization of cyst with tube inserted for drainage.

largely to the head of the pancreas. The main indication for treatment under these circumstances, as mentioned previously, is to relieve pain. Bilateral section of the splanchnic nerves has been used for this purpose by our neurosurgical colleagues. Others have employed somewhat more extensive neurosurgical procedures. Although some brilliant results have been obtained with these operations for relief of pain of pancreatic origin, this has not always been true. Unfortunately some patients are relieved for a time and then experience recurrence of pain. Substitution therapy for pancreatic insufficiency is indicated and is a great aid in improving the patient's general condition. This means that diabetes if present must be recognized and adequately treated. Likewise, steatorrhea requires proper dietary management and the use of large amounts of pancreatin.

COMPLICATIONS OF PANCREATITIS

Many complications may result from recurring pancreatitis. These include pseudocyst of the pancreas, peripancreatic abscess, jaundice, ulceration of the stomach, duodenum or colon, gastrointestinal bleeding, calcareous material in the pancreatic ducts as well as in the parenchyma of the gland and pancreatic insufficiency.

A pseudocyst of the pancreas should be suspected if a mass develops in the upper part of the abdomen of a patient who has recurring pancreatitis (Figure 2). This mass may be transitory in nature and noted only during acute attacks of pain or it may persist, varying somewhat in size from time to time. A pseudocyst of the pancreas is differentiated from a true cyst in that it has no epithelial lining. In our opinion it is best treated by incision and marsupialization (Figure 3). By this means prolonged drainage of the cyst may be provided and this ordinarily results in obliteration of the cyst, provided drainage is established for a sufficient period of time, usually a matter of months. Simple incision and drainage may be employed in the treatment of a relatively small cyst or one that is located in a region which does not lend itself to marsupialization. Complete excision of the cyst is seldom indicated, as ordinarily this requires an extensive surgical procedure which involves unnecessary hazards.

Drainage of a pseudocyst in the pancreas by

anastomosis of the cyst with the upper part of the small intestine has been reported in isolated instances, but is not recommended as a routine procedure, because it may result in stasis of intestinal contents within the cyst or obliteration of the stoma provided for drainage as the cyst decreases in size, before drainage of sufficient duration has been established. Occasionally after a pseudocyst of the pancreas has been drained, the fistulous tract will persist and drainage of pancreatic juice will continue. If this occurs some further surgical procedure may be required. If the fistulous tract becomes well established and is perhaps lined with squamous epithelium, it may be transplanted into the stomach. If this is not the case, however, the tract may need to be excised along with a portion of the pancreas.

Occasionally if definite infection is present a peripancreatic abscess may develop. Commonly this requires drainage. Jaundice may result either from compression of the common duct incident to a pseudocyst, enlargement and induration of the pancreas or an associated stone in the common duct. At times ulceration may occur in the stomach, duodenum or colon secondary to an acute inflammatory process in the pancreas. Profuse hemorrhage from the stomach and duodenum has been observed secondary to acute pancreatitis. Remington and associates[4] have reported 2 cases of stricture of the transverse colon secondary to healing of ulceration in this region which resulted from an episode of acute pancreatitis.

If roentgenologic examination of the pancreas shows calcification within the pancreas it is important to determine if possible whether all of the calcareous material is in the parenchyma of the gland or whether some of it lies within the pancreatic ducts. The term "pancreatic lithiasis" has been used to indicate calculi within the pancreatic ducts in contrast with "pancreatic calcification" which is the term commonly employed to describe diffuse calcification throughout the pancreas without stones being present in the ducts. If stones are present within the pancreatic ducts, they should be removed as otherwise they will cause increased pressure in the collecting system of the pancreas which over a period of time will result in progressive destruction of pancreatic tissue.

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LAENNEC—CLINICIAN AND INVENTOR

Not only did Rene Theophile Hyacinthe Laennec (1781-1826) provide classic descriptions of tuberculosis, lobar pneumonia, pleurisy, bronchiectasis, pneumothorax, emphysema and hydatid cysts of the lungs, as well as characterize the diseases now known as *Laennec's cirrhosis and Laennec's thrombus,* but he also made an invaluable contribution to medical practice with his invention of the stethoscope.

Although auscultation by direct application of the ear to the patient's chest had long been employed, no one before Laennec had devised a simple method for amplifying the heart and chest sounds.

As described by the inventor, the first stethoscope was a "cylinder of wood, an inch and a half in diameter and a foot long, perforated by a bore three lines wide and hollowed out into funnel shape at one of its extremities."

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Highlights in Medical Ophthalmology

THE SCHNEIDER FOUNDATION EYE PRESENTATION

CONRAD BERENS*

NEW YORK UNIVERSITY-BELLEVUE MEDICAL CENTER, NEW YORK CITY

Important eye symptoms in diagnosis of eye conditions having general causes are discussed. Eye lesions in diseases of the kidney, thyroid gland and blood vessels and infections are described. A closer alliance between the general physician and specialist with particular emphasis on the early diagnosis of glaucoma is stressed in the interest of preventing unnecessary blindness.

THE physician, surgeon or specialist who can interpret ocular symptoms and signs adequately has a valuable aid in the accurate diagnosis of many diseases which will insure better and more specific treatment. This paper has been written in the hope that physicians will be aided in making their diagnoses by describing some of the relationships of the eye with remote and adjacent diseased organs and tissues.

Experience has indicated that the history is sometimes of greater importance than the examination in making a complete diagnosis; therefore, some of the symptoms in the field of ophthalmic medicine will be discussed.

IMPORTANT EYE SYMPTOMS IN DIAGNOSIS OF EYE CONDITIONS HAVING GENERAL CAUSES

Head pain and eye pain—Eyestrain is the most common cause of headache due to ocular pathology. Refractive errors, presbyopia, muscle imbalance and aniseikonia are the most common causes of eyestrain. The headache of eyestrain is usually frontal, temporal, between the eyes or behind the eyes and may be accompanied by eye pain. Occipital pain and discomfort are often complained of in eye muscle imbalance. The headache due to refractive errors and ocular motor anomalies usually begins in the afternoon or after performing exacting visual tasks in contradistinction to sinus headache which is usually worse in the morning and tends to improve as nasal drainage improves. Chronic headache is frequently caused by eyestrain, migraine, sinusitis, hypertension, syphilis, brain tumor, glaucoma, herpes zoster ophthalmicus and supraorbital neuralgia. If head pain is one-sided, glaucoma, which often causes blindness, should always be ruled out by determining the tension of the eyeball. An inexpensive hypertension indicator is now available.[1] Ocular neuroses may simulate real ocular pathology but careful elimination of all causes is indicated before the headache is placed on a neurotic basis.

Photophobia—Photophobia or exaggerated hypersensitiveness to light occurs in cases of albinism, foreign body in the cornea, insufficient or poorly sustained accommodation, and inflammation of the eye (conjunctivitis, ulcerative, superficial punctate, interstitial and phlyctenular keratitis; iritis and chronic cyclitis). Patients with arthritis and chronic sinusitis often com-

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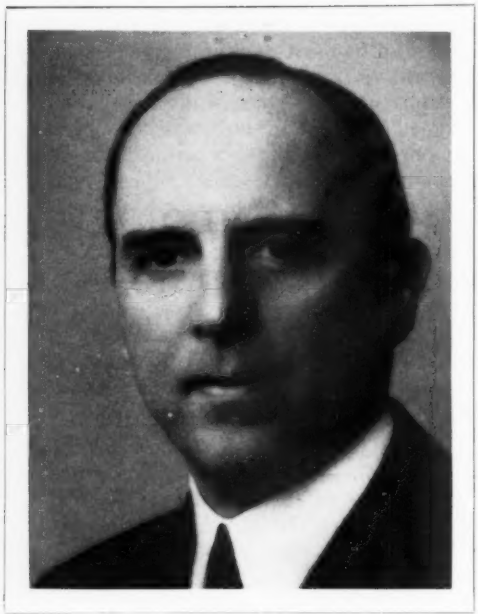

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plain bitterly of photophobia. Photophobia may be a symptom of meningitis, riboflavin deficiency and hysteria.

Lacrimation and epiphora—Lacrimation or excessive normal secretion may result from the presence of a foreign body in the cornea or in the tarsal conjunctiva, trichiasis, distichiasis, meibomianitis, chronic conjunctivitis, allergy (for example—hair dye, face powder), keratitis, iritis and chronic nasal infection causing irritation of the fifth nerve. Local or general factors which irritate the nerves supplying the lacrimal glands may cause lacrimation. Increased intraocular pressure, imbalance of the extra-ocular muscles, and reading without correction of hyperopic or astigmatic errors of refraction may also produce tearing. Blocking of one or both puncta or of the nasolacrimal duct by inflammation, fungi, or a cilium and infection of the lacrimal sac (dacryocystitis) and ectropion with eversion of the punctum may result in overflowing of the tears (epiphora).

Diplopia—Rule out normal physiologic diplopia by asking whether sight becomes confused, not whether the patient has double vision. Monocular diplopia may result from diffusion circles in errors of refraction, especially in astigmatism, which produces a double outline of one image rather than two distinct images of the object observed; from a double pupil in single or multiple tears of the iris and occasionally in iridocyclitis; from subluxation, partial cataract or sclerosis of the lens.

Binocular diplopia may result from horizontal or vertical imbalance or paralysis of the extraocular muscles (syphilis, encephalitis, brucellosis, convergence weakness in sinus disease), anisometropia, displacement of the eyeballs, general diseases and from toxemias. In some general diseases, such as encephalitis lethargica, multiple sclerosis, syphilis, diabetes and arteriosclerosis; in toxemias developing from poisoning (foods or gas) and following cranial trauma, diplopia may occur suddenly as a result of paralysis of the ocular neuromuscular mechanism. It is important to ascertain whether there has been a previous attack of diplopia. This may be suggestive, in a young person, of disseminated sclerosis, encephalitis or intracranial neoplasm, and in an older person of syphilis, arteriosclerosis or diabetes.

Congestion of the eyelid borders—Redness associated with congestion or inflammation of the margins of the eyelids is frequently caused by: (1) contact dermatitis, which results from direct contact with drugs, cosmetics or plants, and is accompanied by scaling and itching of the skin of the eyelid (the supratarsal fold of skin is usually involved); (2) allergic dermatitis resulting from allergy to drugs, foods, pollens, animal danders or the bacteria of focal infections; (3) recurrent attacks of styes (hordeola) resulting from suppuration in a gland of Zeis and chalazia caused by inflammation of the meibomian glands (Figure 1). Styes are characterized by redness and edema of the eyelid followed by the appearance of a localized yellowish spot at the root of a cilium on the skin surface of the eyelid. A sty usually breaks through the skin and discharges pus. One or more cilia may be affected. Associated nasal or sinus infection should be ruled out in chronic or recurrent cases of inflammation of the eyelids.

Failing distant vision—Indistinct, blurred or obscured distant vision in children and in adults may be caused by uncorrected hyperopia and astigmatism, uncorrected or increasing myopia, cataract, degeneration of the macula (suspect if central vision is mainly affected), nystagmus of congenital origin or the toxic action of chemicals (quinine, lead, carbon disulfide) or bacteria on the retina and optic nerve as well as by the toxic effect of tobacco and wood alcohol.

Failing near vision—The sudden onset of poor near vision in patients under 42 years of age is suggestive of acute hyperglycemia (diabetes), involvement of the ciliary body (cyclitis), glaucoma

and sympathetic ophthalmitis if one eye has been injured. Presbyopia is the most common cause of failing near vision after 40.

Colored rings around lights—A halo or ring of rainbow colors may be seen around artificial lights in primary glaucoma and in secondary glaucoma associated with cyclitis. Chronic infections and psychologic disturbances should be eliminated as possible etiologic factors.

Nyctalopia or night blindness—This may occur in patients who have a central scotoma associated with amblyopia resulting from tobacco, alcohol or other toxins (toxic amblyopia), or it may be associated with disease of the macula lutea Deficiency in vitamin A causes impaired night vision only in rare instances except under famine conditions. The most common ocular cause is retinitis pigmentosa.

Metamorphopsia or distorted vision—This condition usually is caused by errors of refraction, especially astigmatism, or to lesions in the macular area of the retina or the choroid underlying the retina. If the condition persists rule out cerebellar and labyrinthine disturbances.

Macropsia—Objects appear larger with one eye than with the other. This condition results when the retinal elements (cones) are crowded together in a smaller area than they normally occupy causing the stimulation of more cones than the object observed would normally stimulate; and in paralysis of accommodation associated with dilation of the pupil, and in aniseikonia.

Micropsia—Objects appear smaller with one eye than with the other. Retinal elements in these cases are spread apart, as in acute retinitis, or in the presence of spasm of accommodation associated with contraction of the pupil, also in anisometropia, antimetropia and aniseikonia.

Chromatopsia—When objects appear abnormally colored, for example when snow seems yellow (xanthopsia), often experienced by a jaundiced patient or one poisoned by santonin, the condition is termed chromatopsia. In digitalis poisoning and occasionally in diseases of the choroid and retina, green, blue or violet vision may be noted. At times objects may appear red or blue after the removal of a cataract.

Visual field disturbances—Although visual field impairment produced by bilateral central cataract and macular degeneration may be noted by the patient, large unilateral field defects and even blindness of one eye may go unnoticed for some time especially if the sighting or dominant eye is not affected. Right lateral homonymous hemianopia (cerebral arterial occlusion, brain hemorrhage, brain tumors) may cause little disturbance even in reading but the left defects are usually bitterly complained of. Quadrant defects (often vascular) and small paracentral scotomas (glaucoma and lesions of the choroid and retina) usually require careful perimetry for detection. The bitemporal hemianopias of pituitary lesions are usually recognized earlier than the nasal field losses of chronic noncongestive glaucoma. The central scotomas and the bilateral hemianopic (lateral and vertical) transient visual disturbances of migraine are usually accompanied by zigzag shimmering semicircular lines of light. Visual fields should be plotted to rule out organic lesions of the brain.

FIGURE 1. Hordeolum on the lower eyelid and chalazion affecting the upper eyelid.

HEREDITARY EYE DISEASES AND CONGENITAL ANOMALIES

Congenital color amblyopia—Congenital so-called color blindness is a hereditary, familial, sex-linked and frequently recessive anomaly of color vision. Although it may be associated with other lesions as retinitis pigmentosa and macular degeneration, eyes so affected are usually normal. Anomalous perception of red and green is often undiagnosed and is important because of the use of red and green in transportation, military signals and industry. Approximately 4 per cent of males and 0.5 per cent of females have some form of anomalous color perception. There is no cure for this anomaly[2] in spite of some reports[3] to the contrary.

Retinal hemorrhages—These may be congenital and are often found in the newborn. They are especially common (41 per cent) in immature and premature infants.[4] Amblyopia and nystagmus are sometimes attributed to this cause.

Medullated nerve fibers—Grayish white patches with feathery edges may develop shortly after birth at the border of the optic nerve or in other areas of the retina. This is a developmental, not truly a congenital anomaly and does not impair vision.

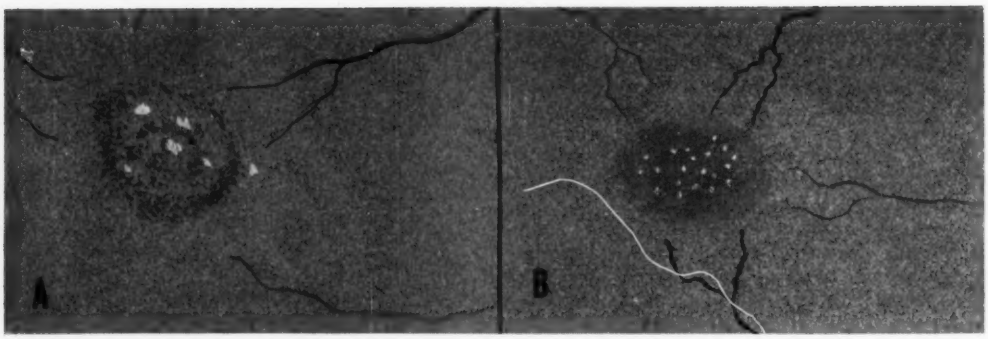


FIGURE 2. Macular degeneration: (A) juvenile type; (B) senile type.

Pigmentation of the retina in groups—Small dark brown patches of pigment may be found in the retina.[5] Pigmentation of the retina is usually unilateral and is more common in males than in females It is not hereditary, is usually associated with other anomalies of structure and function is unimpaired.

Retinosis and night blindness with and without pigment—The diagnosis of the pigmented type (retinosis or retinitis pigmentosa) is made on several findings. The pigment which often invades the sheaths of the vessels characteristically appears like bone corpuscles, first forms in the periphery of the retina and gradually encroaches on the nerve and macular area. The association of night blindness and increasing constriction of the visual fields often with a ring scotoma makes the diagnosis certain. The only difference between the pigmented and nonpigmented type is the absence of pigment and both types are classified as tapetoretinal degenerations. As the disease progresses, the retinal vessels become more and more constricted and the nerves become atrophic, assuming a grayish waxy color. Treatment seems to be largely unsatisfactory although diathermy, cervical sympathectomy, injections of cod liver oil and tissue therapy have been tried. The cataracts (usually posterior cortical) which often develop may be removed if sufficient visual field or vision remains.

PIGMENTARY retinosis may be associated with hypogenitalism, obesity of the Fröhlich type and retarded mental development, the Laurence-Moon-Biedl syndrome. The use of thyroid and pituitary extracts may be beneficial.

Retinosis (retinitis) punctata albescens—This condition is allied to pigmentary retinosis but there is no pigment, only small well defined grayish white dots scattered through the fundi. The condition is hereditary, vision is gradually impaired and treatment is unavailing.

Amaurotic familial idiocy (Tay-Sachs disease)—This is a hereditary familial degeneration of the ganglion cells of the eye, brain and spinal cord caused by deposits of lipoids in the cells. The macular region exhibits a circular white area with a red central spot.

Macular degeneration (certain types)—The times in years for the development of macular degeneration are at birth (congenital type); from 6 to 8 years of age (infantile type); between 14 and 20 years (juvenile type); from 50 years on (beginning of senile involvement) and the senile type.

Symptoms in the infantile type consist of small light gray spots increasing slowly in definition changing to orange or dirty yellow in color. Later, scattered between the yellow spots or yellow background, fine round pigment spots develop, covering the entire macular area uniformly (Figure 2a). In the advanced stages the spots coalesce and form a large, round or oval, dirty yellow patch; pigment dots or larger clumps usually appear and increase with age.

The presenile and senile types (macular or central chorioretinitis) usually show, upon the dark brown color of the macula, numerous yellow spots with indistinct or well defined margins (Figure 2b). At times the macula is discolored showing a reddish yellow background with large light yellow spots with indistinct margins and black pigment spots interspaced.

Although the lesions are usually bilateral, senile degeneration may be confined to one eye for a long period of time. In some cases the sclerosis of the choriocapillaris produces, from the beginning, a complete depigmentation of the pigment epithelium and the net of the choroidal vessels appears clearly at the macula, sometimes showing white lines of sclerotic degeneration.

Another type of senile degeneration is characterized by the development of colloid deposits in the lamina vitrea (Drusen)—small round yellowish white spots form around the disk and in the

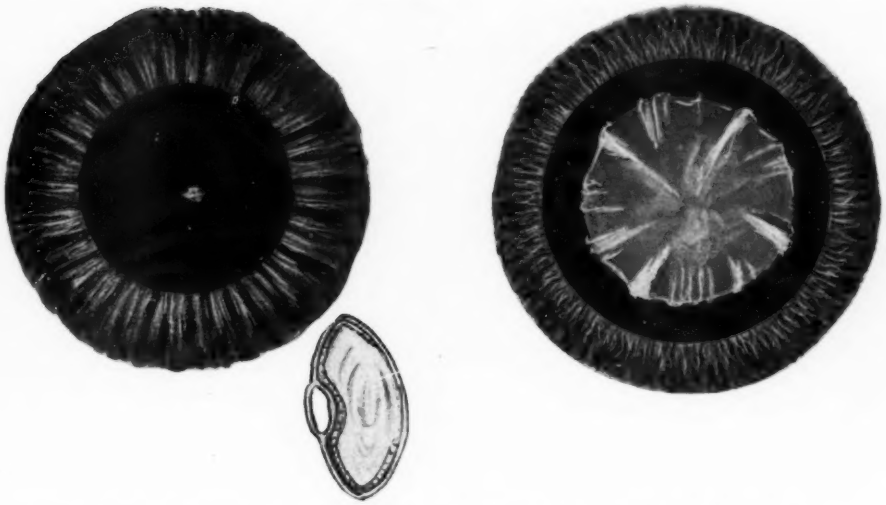


FIGURE 3. Forms of cataract: (A) *(left)* anterior polar cataract; (B) *(right)* zonular cataract.

macula. The spots are colloid deposits and are probably produced by the pigment epithelium.

In the last stages of the juvenile form, the papillomacular bundle is damaged and a temporal atrophy, which rarely becomes complete, develops in the disk.

The functional symptoms may precede the ophthalmoscopic lesions. There is a slow but progressive impairment of central vision, especially in daylight, and a relative scotoma for colors. Later, an absolute scotoma appears and the child forms the habit of looking sidewise. The color sense is impaired, light adaptation is disturbed and hemeralopia develops. In the presenile and senile types, central vision fails gradually and often positive scotomas appear near the point of fixation. Usually, one eye is affected before the other. Metamorphopsia develops later and varies in degree according to the retinal changes.

The infantile type is usually associated with cerebral degeneration and imbecility, while in the juvenile form the eye alone is affected in the majority of cases. In the latter, the disease may remain stationary but, if associated with mental deterioration, it may increase progressively and epileptic seizures often appear followed by complete idiocy lasting until death ensues (progressive familial macular degeneration).

LENS

CATARACT may be congenital or hereditary.

Congenital cataracts—These usually result from anomalies of development of the lens and the most commonly encountered forms are the anterior and posterior polar types (Figure 3a). In these cases dot-like opacities may be seen at the anterior or posterior pole of the lens. Those at the posterior pole are considered as remains of the hyaloid artery. Neither of the opacities interferes with vision and no treatment is required. Zonular (Figure 3b) or lamellar cataract is observed as a grayish disk-like opacity in the center of the lens. If vision cannot be improved with lenses, optical iridectomy or needling of the lens is indicated.

Hereditary cataract—This anomaly is usually sex-linked and tends to appear earlier in succeeding generations but families are not affected as to longevity, fertility or health (Figure 4).

Congenital aphakia—Absence of the lens is rare.

Lenticonus—A cone-shaped area may protrude from the anterior or posterior surface of the lens (Figure 5) and usually disturbs vision because of high, irregular myopic astigmatism. Careful refraction is the best treatment.

Coloboma—A notch or coloboma in the lens is often associated with coloboma of the iris, choroid or eyelid.

Dislocation—Both lenses are usually displaced nasally, a congenital anomaly which may be hereditary. In slight dislocation, careful refraction may suffice but needling or total extraction of the lens is often required. Marfan's syndrome (1896) is the name applied to bilateral congenital and hereditary dislocation of the lenses which later is associated with unusually long bones of the leg, arms and fingers (Figure 6). The condition is hereditary and familial, affecting males more than females.[6]

Retrolental fibroplasia and prematurity (Figure

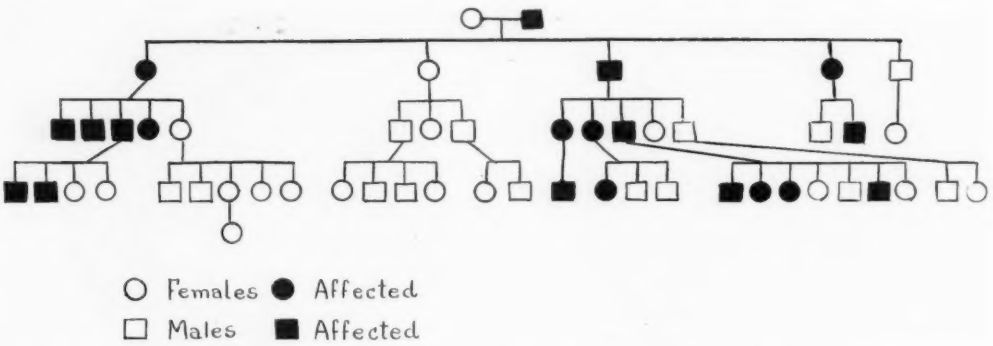


FIGURE 4. Pedigree of hereditary familial cataract affecting five generations. Family of 52 persons, 20 individuals affected with hereditary cataract.

7)—This disease is usually associated with prematurity and usually affects both eyes. The incidence has been found greater in the 3 to 4-pound premature weight group. The most conspicuous diagnostic sign is the presence of a membrane, often vascularized, situated behind the crystalline lens. The presence of blood vessels in the membrane serves to differentiate retrolental fibroplasia from congenital cataract. Secondary changes, such as synechias, glaucoma and iritis, frequently develop. Preventive and curative treatment have been disappointing.

Toxoplasmosis (Figure 8)—Prenatal infection (transplacental) of the eyes of human infants by *Toxoplasma* has been reported with increasing frequency in the past 10 years. It is a protozoon now considered to be an obligate intracellular parasite. Toxoplasmosis may cause the following anomalies: (1) congenital detachment of the retina; (2) microphthalmos; (3) congenital cataract; (4) "coloboma" of the macula; (5) congenital total detachment (pseudoglioma), bilateral and frequently multiple chorioretinitic lesions are common and of diagnostic importance. Macular lesions are common and usually associated with nystagmus. Any child showing one or several of these symptoms should be thoroughly examined for toxoplasmosis by the neutralization test in the blood, x-ray of the skull (scattered areas of calcification) and examination of the cerebrospinal fluid.

Congenital cataract and rubella (German measles)—Congenital cataracts have been found by Gregg[7] and others in many infants whose mothers had rubella especially in the first, second and up to the third month of pregnancy. Pigmentation of the retina described by Gregg (1939) as "blotchy" and unlike the bone-corpuscle picture of typical retinitis pigmentosa, as the macular region is most affected, was encountered in 50 per cent of the deaf children studied by Marke.[8a]

OPTIC NERVE

Optic atrophy (Leber's disease) is usually detected in the second decade and may exhibit sex-linked dominance.[8]

Congenital holes and colobomas of the optic nerve disk have been reported.

Errors of refraction—Myopia, hypermetropia, astigmatism. There is no question that myopia and other errors of refraction may be hereditary and often familial. The work of Brown[9] supports the biologic and hereditary etiology of myopia. The influence of heredity was proved and that of close work disproved by Jancke[10] in his studies of 150 unselected pairs of twins.

EYELIDS

Ptosis may be unilateral or bilateral (Figure 9). It is usually caused by faulty development of the levator, and other extraocular muscles are usually affected. In jaw winking there is unilateral ptosis and the eyelid is raised by opening the mouth or moving the jaw to one side.

Congenital coloboma of upper or lower eyelid (Figure 9).

Epicanthus or a vertical fold of skin on either side of the bridge of the nose (Figure 10) is a common anomaly and is also seen in Mongolian idiocy. Plastic surgery is indicated if the normal development does not mask the deformity.

OCULAR MUSCLES

Ocular muscles may be affected by anomalies of structure, origin, insertion or innervation.

The superior rectus muscle is commonly affected and often associated with ptosis. The lateral rectus muscle is less commonly affected and in one case[11] was associated with "status thymicolymphaticus."

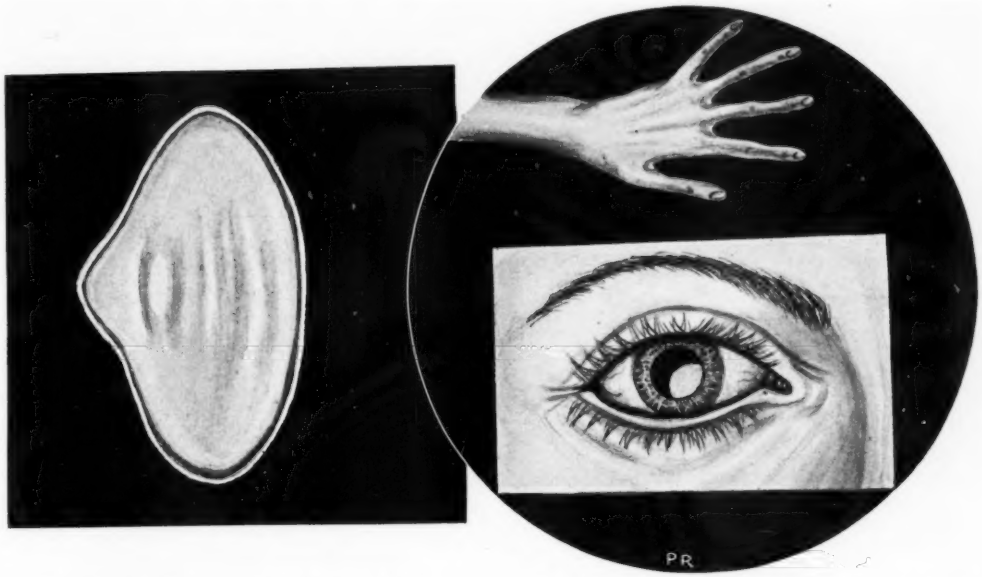


FIGURE 5 *(left)*. Lenticonus. Note the cone shaped protrusion of the anterior surface of the lens. FIGURE 6 *(right)*. Subluxation of the lens in arachnodactyly. Note the eccentric position of the lens in the pupil, and the unusually long slender fingers.

EYEBALL

Glaucoma is sometimes found as a familial tendency. Glaucoma may develop in hereditary lesions with marked aniridia where only a peripheral collar of iris tissue persists.

Buphthalmos (hydrophthalmos), synonym infantile glaucoma. The eyeball and cornea are large and the tension is elevated. It is often unilateral and both sexes are affected equally.

Megalophthalmos—Enlargement of the eyeball is usually bilateral and there is no hypertension except as a complication. Males are usually affected.

ALBINISM

The pigment is not only absent from the hair and eyelashes (Figure 11) but vision is often seriously disturbed because of lack of pigment in the retina which may be partial and merely central (Figure 12). Vision is reduced because of the absence of visual purple.

IRIS

Aniridia—In this condition the iris muscles are absent and only a narrow collar of tissue is found. Other congenital and hereditary anomalies may be associated such as cataract and glaucoma.

Congenital coloboma—Part of the iris is absent, usually below, and coloboma of the choroid, ciliary body and lens may be associated.

Persistent pupillary membrane—Single or multiple strands usually extend between areas of the lesser circle of the iris or from the lesser circle to the lens capsule.

VASCULAR DISEASES AND RETINAL CHANGES; CLASSIFICATION OF RETINAL LESIONS IN THE PRESENCE OF VASCULAR HYPERTENSION[12]

As basic principles in the study of the retinal complications of cardiovascular renal disease with hypertension, it may be stated that:

1. The ophthalmoscopically visible arterial vessels of the retina should be designated as arterioles.

2. By the term "retinopathy" in hypertensive or renal disease is meant the (usually) abruptly developing involvement of the retina characterized by serous and hemorrhagic extravasations into and under the retina. This serous extravasation may be represented by diffuse edema, by localized "cotton-wool" patches, or by the more chronic punctate deposits considered to be the late results of residues of edema and which include "macular star" formation. At times, edema of the nerve head occurs in association with the retinopathy. For this combination, the terms "neuroretinopathy" or "retinopathy with papilledema" are acceptable.

3. The component elements of "retinopathy" are

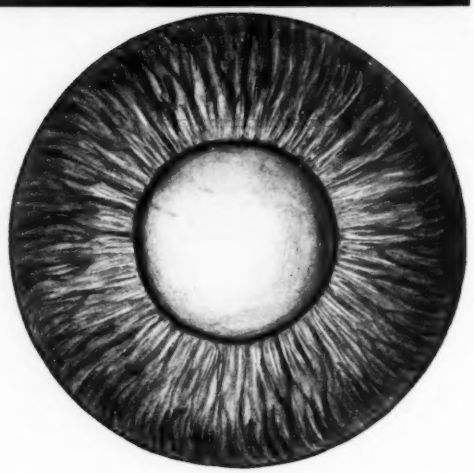
FIGURE 7. Retrolental fibroplasia: *(left)* Note the papilledema and the gray exudative mass in the retina in the infiltrative stage of retrolental fibroplasia. *(right)* Note the vessels extending from the disk to infiltrated retina which gives the appearance of an unusual detachment. Note the dense membrane back of the lens in which blood vessels may be seen.

basically identical in all forms of hypertensive and renal disease.

4. "Grading" of the severity of the arteriolar lesions is of value, particularly in certain situations, such as when decision is necessary as to the advisability of surgical treatment, and when it is desired to evaluate progression of the lesions, but grading is not essential ordinarily for proper identification of the types of the lesions.

5. Elevation of blood pressure in itself is the most essential factor in production of the arteriolar and retinal lesions, although constriction of the systemic arterioles in response to some circulating vasopressor substance, or to a central reflex mechanism, may be a factor in initiating the rise of blood pressure. Therefore, practically identical lesions can occur both in primary essential hypertension, and in hypertension secondary to acute or chronic glomerulonephritis, toxemia of pregnancy, unilateral atrophic kidney, pyelonephritis, pituitary basophilism, adrenal tumor, periarteritis nodosa, and the like.

It is suggested that the terms "arteriosclerotic retinopathy" and "arteriolosclerotic retinopathy" be dropped (or used only as last resorts), since the lesions usually so designated (punctate hemorrhages and punctate white areas) are likely to be end results either of venous occlusion or hypertensive retinopathy and at times they are of diabetic origin.



For the diagnosis of retinopathy, the presence of either cottonwool patches or edema residues is requisite.

CLASSIFICATION OF ANGIOSPASTIC CENTRAL RETINOPATHY WITH LITTLE OR NO HYPERTENSION

According to Marquardt[13] the angiospastic hypotensive cases with central angiospastic retinopathy may now be divided into four groups: (1) Metabolic disorders, especially hypothyroidism and including gout; (2) Chemical electrolyte disturbances and particularly subclinical tetany; (3) Neurogenic group (neurocirculatory asthenia and adolescent vascular instabilities) and (4) Allergic group; probably thermal allergy may be most important because most cases seem to be precipitated by exposure to cold weather or chilling.

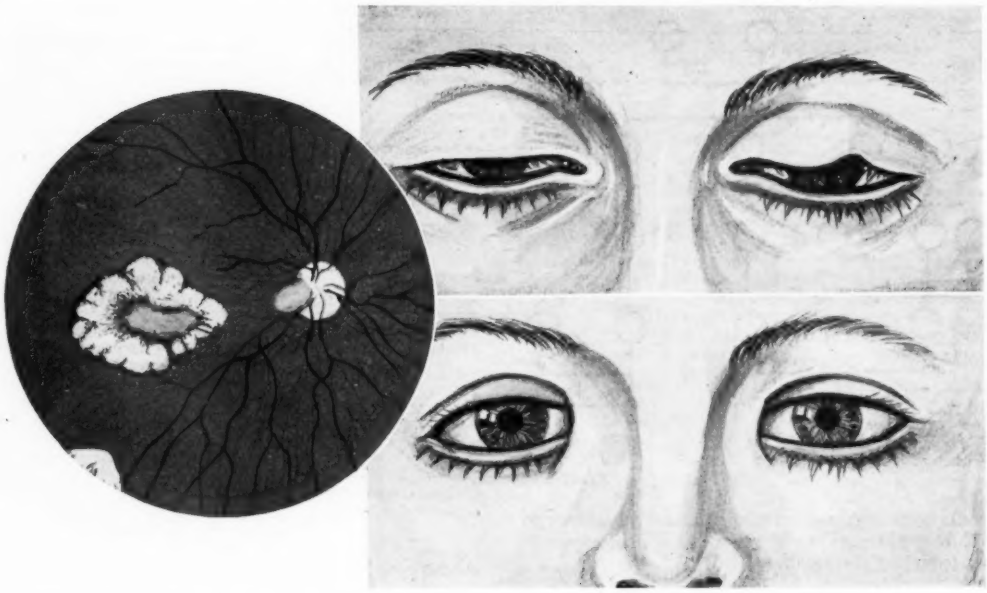


FIGURE 8 *(left)*. Toxoplasmosis. Note old chorioretinal lesions, one in the macula and one in the periphery. The disk shows a slightly elevated lesion. FIGURE 9 *(upper right)*. Bilateral ptosis and coloboma of the left upper eyelid. FIGURE 10 *(lower right)*. Epicanthus. Note the vertical folds of skin, covering the medial canthi.

TREATMENT OF HYPERTENSIVE RETINOPATHY

There is no question that patients seen by me have been greatly benefited by sympathectomy but unfortunately many patients are seen too late for this form of treatment and usually have seriously impaired vision. Recently Marquardt[13a] and Page[13b] have had excellent results in advanced hypertensive retinopathy with a nonprotein bacterial pyrogen (Pyromen R), which produces fever with much less discomfort than other foreign protein. Even in these advanced cases the rice diet as administered by Kempner and advocated by Anderson[14] has produced striking results from the eye and general points of view.

Although patients complain of the rice diet, the results I have observed amply justify the use of this form of therapy, which is so much less drastic and dangerous than sympathectomy.

DISEASES OF THE KIDNEYS

In the production of retinal changes (renal retinopathy) two factors are essential, namely generalized vasoconstriction (arterial) and hypertension. Therefore retinal lesions may be absent in lipoid and amyloid nephrosis, embolic and nonembolic glomerulonephritis and in uncomplicated arteriosclerosis of the retinal artery and its larger branches.

Retinal changes (renal retinopathy) in progressive diffuse glomerulonephritis include (1) diffuse arteriolar constriction; (2) edema of the retina especially around the disk and over the disk (papilledema), and retinal hemorrhages (usually linear or flame-shaped and usually near the vessels); (3) white spots with sharp outlines, cottonwool patches and star-shaped figure around the macula, all of which are the remains of edema. Wagener[15] believes that it is difficult to attribute these changes to any particular mechanism but that they are basically nutritional is not doubted. Chronic diffuse glomerulonephritis will produce retinopathy if constriction of the retinal vessels is present and sclerotic changes in the retinal arteries are usually found in the later stages of the disease. Retinal detachment may be a serious late complication.

Nephrosis—That of pregnancy is most important and if there is continued generalized arterial constriction hypertension, retinopathy will develop and produce a picture similar to that caused by acute diffuse nephritis.

Retina in arteriosclerotic diseases of the kidney—No changes may be seen in senile arteriosclerosis. In essential hypertension no changes may be noted but later arteriolar sclerosis may be observed in the retina, concurrently with the development of cerebral arteriosclerosis. Later if sudden generalized

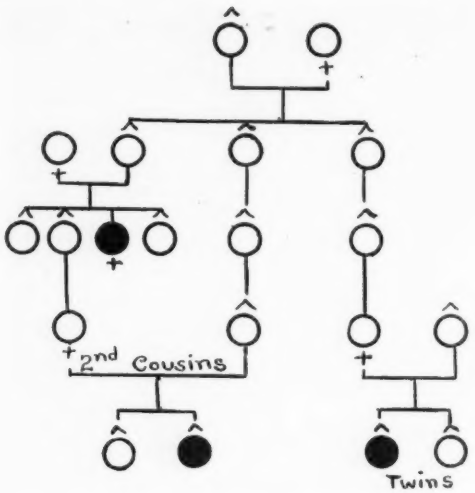


FIGURE 11. Pedigree illustrating recessive inheritance in albinism. The presence of genes in both chromosomes is essential for the manifestation.

(After Anderson, J. R., in Berens, C., "The Eye and Its Diseases," W. B. Saunders, Ed. 2, 1950.)

constriction of the arteries occurs, the retina may exhibit signs of malignant hypertension or malignant renal sclerosis. These changes are those of diffuse nephritis with marked arteriosclerosis; even obliteration of some branches and marked edema (5 to 6 D) of the disk.

CLASSIFICATION OF VASCULAR HYPERTENSION

TERMINOLOGY	DIAGNOSTIC LESIONS IN RETINA
Neurogenic hypertension	None, even in cases of considerable duration, except mild generalized narrowing of arterioles
Acute hypertension ("angiospastic")	No arteriolosclerosis. Generalized narrowing of arterioles. Focal constriction in arterioles. Edema of retina, cottonwool patches, hemorrhages (usually). Edema of disk (often). May terminate in recovery, in chronic hypertension or in rapidly progressive (fulminating) malignant hypertension
Chronic nonprogressive ("benign") hypertension	Only mild generalized narrowing of arterioles (grade 1 or 2) if seen in early years. Also mild generalized arteriolosclerosis (grade 1 or 2) if seen after several years' duration of the hypertension. At times ischemic or hemorrhagic infarctions of the retina
Chronic progressive hypertension	Always generalized arteriolosclerosis. Generalized narrowing of arterioles. Focal constrictions in arterioles. At times focal arteriolosclerosis, cottonwool patches and hemorrhages in the retina
Terminal malignant hypertension	Always generalized arteriolosclerosis. Always papilledema. Generalized narrowing of arterioles. Focal constrictions in arteriolosclerosis. Usually edema of retina, cottonwool patches, hemorrhages, often residues of edema in form of macular stars or scattered, punctate deposits

N.B. Any of these varieties or phases of hypertension, except the neurogenic variety, may be primary ("essential") or may be secondary to some demonstrable causative lesion or disease.

DIABETES

Ocular signs of diabetes may include paresis or paralysis of the ocular muscles, iritis, alterations in refraction, snowflake cataract (Figure 13), and retinopathy (exudates and superficial, and deep hemorrhages).

Retinopathy—When retinopathy or retinosis develops in diabetes, raised blood pressure and vascular diseases are often but not always present, and albumin as well as sugar is commonly found in the urine; retinopathy seldom occurs in young subjects suffering from the worst form of the disease. The duration of diabetes is an important factor in the development of retinopathy and is more important than age.

In regard to the foregoing, it is imperative to see whether there are reasons for identifying a condition to which the term "diabetic retinopathy" can be properly applied. It is clear that there is a similarity about renal, hypertensive and diabetic retinopathy, and it is by no means always possible to say, from ophthalmoscopic appearances alone, which of these three is present. There is, however, no doubt that in many cases appearances are so characteristic that one is able to identify the type of retinopathy with assurance.

Retinal changes in diabetes include yellow, waxy, somewhat glistening, usually sharply circumscribed patches, often arranged around the macula (lipoid and hyaline deposits). Hemorrhages are usually rounded and have clearcut edges quite unlike the more superficial flame-shaped hemorrhages of renal retinopathy. The rounded hemorrhages are difficult to differentiate from the microaneurysms which are quite typical of diabetic retinopathy and Ballentyne demonstrated years ago that aneurysms were frequently associated with the hemorrhages. According to Elwyn,[16] the hemorrhages are caused primarily by a state of prestasis in the terminal vessels induced by prolonged hyperglycemia with diapedesis and hemorrhages leading to deficient oxygenation and nutrition of the retina. Arterial and toxic or biochemical changes of diabetes are causal.

GRADING OF LESIONS OF RETINAL ARTERIOLES

GENERALIZED NARROWING OF ARTERIOLES	FOCAL CONSTRICTION OR FOCAL SCLEROSIS OF ARTERIOLES	GENERALIZED SCLEROSIS OF ARTERIOLES
Grade 1 Reduction of caliber of arterioles to 3/4 average caliber or 1/2 caliber of veins	Localized narrowing to 2/3 caliber of proximal segment of arteriole	Brightening or increased luster of arterioles, mild depression of veins at the points of arteriolar crossings with reduction of visibility of those portions of veins which underlie the crossing arterioles
Grade 2 Reduction of caliber of arterioles to 1/2 average caliber or 1/3 caliber of veins	Localized narrowing to 1/2 caliber of proximal segment of arteriole	Burnished coppery color of the arterioles, with definite depression of underlying veins, widening of apparent arteriolovenous crossing spaces and almost complete invisibility of those portions of veins which underlie the crossing arterioles
Grade 3 Reduction of caliber of arterioles to 1/3 average caliber or 1/4 caliber of veins	Localized narrowing to 1/3 caliber of proximal segment of arteriole	Polished silver color of arterioles, widening of apparent arteriolovenous crossing spaces with a change in the course of veins ("right-angled crossings"), complete invisibility of those portions of veins which underlie the crossing arterioles and distal dilatation of the veins
Grade 4 Arterioles thread-like or invisible	Arteriole invisible beyond point of constriction (or in the case of focal sclerosis visible only as a thin, fibrous cord)	Arterioles visible only as fibrous cords, without blood stream

That hypertension is not the cause of diabetic retinopathy or intraocular hemorrhage is suggested by the following studies: In a group of 50 diabetic patients who were operated upon for cataract, 20 had diabetic retinopathy. In this group of 20 patients, who averaged 63 years of age, the blood pressure averaged 165/88. Of the 30 patients without diabetic retinopathy the blood pressure averaged 164/92. Eleven of these 50 patients had hemorrhages following cataract extraction, and their blood pressure averaged 147/82, postoperatively. From these figures it would be difficult to implicate hypertension as the cause of the retinopathy or the retinal hemorrhages.[17]

It is my opinion that there is a retinopathy proper to diabetes and which therefore is properly named "diabetic retinopathy." Retinal changes may be discovered at a time when the patient's urine is sugar-free, and in the presence of hypoglycemia. This assumption is strengthened by the findings of similar pathology in young patients by O'Brien and Allen.[18]

Retinopathy may develop when the disease is apparently controlled by insulin and although certain edema residues may disappear, it is unlikely that retinopathy is cured by this treatment. Nor do I believe that insulin intake or the level of the blood sugar is closely related to the development of retinal hemorrhages. The value of vitamin P and vitamin C (rutin, rutorbin) in controlling retinal hemorrhages is not proved.

Prognosis—The presence of retinopathy in diabetes is an indication of some gravity but it is by no means so serious a sign as retinopathy in renal disease. Thus Nettleship found that, of 49 patients with diabetic retinopathy, 60 per cent lived for more than two years.

Cataract—Opacity of some degree in the lens in persons past middle age is about as common as gray hair, and often is of no greater significance in regard to the patient's general condition. It is clear then, that the term "diabetic cataract" is not appropriate to opacities such as happen to be present in a subject with diabetes.[19]

THE true diabetic cataract occurring in young subjects affects both eyes, begins in the posterior layers of the cortex, and rapidly matures (snowflake cataract) (Figure 13). Diabetes should be controlled if a cataract is to be extracted but complications of operation do not seem to be greatly increased in diabetes.[20]

Retrobulbar neuritis—By this term is meant an inflammation of the extra-ocular part of the optic nerve, caused by toxins, some endogenous and some exogenous; among the endogenous causes, diabetes takes a place.

Diabetics are more susceptible to tobacco amblyopia than are healthy subjects and some have thought that it was this association which was responsible for the signs of retrobulbar neuritis in diabetes, but cases have been reported in patients who used no tobacco.

Retinopathy may or may not be present and in chronic cases some degree of optic atrophy may develop.

Iritis—It is a common experience that the operation for the removal of a cataract in a diabetic is more likely to be followed by a rather troublesome iritis than in healthy individuals, but apart from this, a spontaneous iritis may arise from no apparent cause. If numerous vessels develop in the iris around the pupil, rubeosis is said to be present and glaucoma should be excluded.

Lipemia retinalis—It is doubtful whether lipemia attains a sufficiently high degree (lipoids in blood over 4 per cent) in any condition, other than diabetes, to give rise to the ophthalmoscopic picture. The milky appearance, at first limited to the peripheral vessels, extends toward the disk until, in the fully developed stage, all the vessels, arteries and veins alike look as if they were filled with cream, except perhaps on the disk where a pale cherry color persists. Neither hemorrhages nor exudates occur and vision is not affected.

Paralysis of ocular nerves—In a few cases, paralysis of one or other of the ocular muscles may occur, due apparently to a peripheral neuritis. The lateral rectus muscle is most commonly affected.

Alterations of refraction—Rapid variations in refraction are probably due to changes in the refractive powers of the lens, and probably vary with the blood sugar content; the variations have been as much as 5 D. Hyperopia usually decreases.

Changes in accommodation—Dysaccommodations may develop and stronger lenses for reading may be required.

ENDOCRINE DYSFUNCTION

During the examination of the eyes any evidence of endocrine (thyroid, parathyroid, pancreas, suprarenal, pituitary, pineal, thymus, testes and ovaries) disturbances should be noted in order to facilitate diagnosis. The various manifestations may be summarized as follows:

Eyeball—Exophthalmos and disturbances of ocular motility may be observed in thyrotoxic and thyrotropic types of thyroid-pituitary dysfunction (disease of the thyroid gland).

Refraction—Transitory changes in refraction may occur in diabetes (pancreas). High myopia may possibly result from endocrine dysfunction.

Muscles—Extra-ocular muscles may be affected; Graefe's, Möbius', Gifford's and Stellwag's signs in Graves' disease; convergence and accommodation may be weakened in myasthenia gravis.

Eyelids—Edema, angioneurotic edema and pigmentation may be seen in Addison's disease. Ptosis, which may at first be limited to one side, is the commonest early sign of myasthenia gravis, which may be associated with thymus lesions (Doggart). The diagnosis is assured if PROSTIGMINE relieves the symptoms. Twitching of the orbicularis fibers of the eyelids may occur from deficiency of calcium associated with parathyroid pathology. Puffiness of the eyelids is observed in cretins (congenital) and myxedema (acquired) as the result of hypoplasia of the thyroid gland.

Cornea—Keratoconus, adiposogenital and familial dystrophy of the cornea may be caused by endocrine disturbances.

Sclera—Blue sclera (fragilitas ossium).

Iris—Diabetic iritis (pancreas).

Lens—Cataract and low blood calcium (parathyroid deficiency). Snowflake cataract is seen in diabetes.

Retina—Diabetic retinopathy, lipemia retinalis and retinal hemorrhages in diabetes. Degeneration of the retina and possibly retinitis pigmentosa.

Optic nerve and visual pathways—Chiasmal pressure producing gradual loss of vision and bitemporal hemianopsia in pituitary lesions. Papilledema may be an early sign of a tumor of the pineal gland.

DISEASES OF THE THYROID GLAND

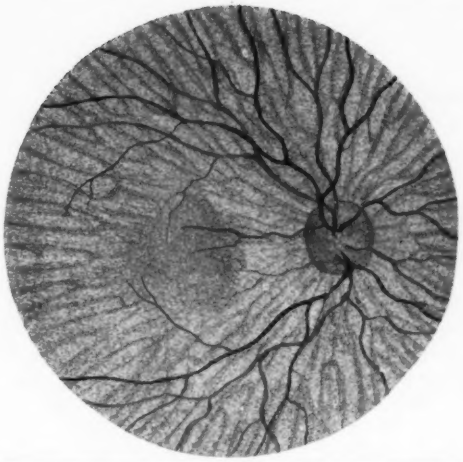
Exophthalmic goiter (Graves' disease), hyperthyroidism—The ocular symptoms of this disease, which is more common in females, are striking and important, and one or other of them is almost invariably present. These are, however, limited to the adnexa of the eyeball; no pupillary or fundus changes occur. Focal infection and/or prolonged nervous tension seem to be factors in some cases and thyroidectomy usually does not cure all the ocular signs.

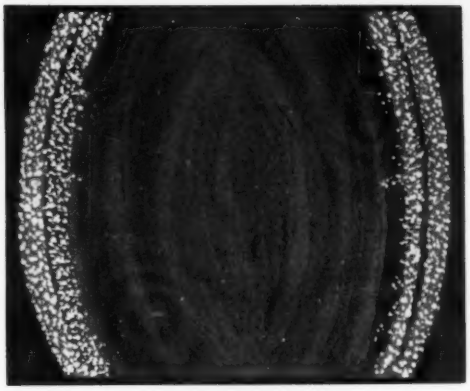
Exophthalmos—In the early stages one eye only may be affected, but the other will later be involved. The increased exposure of the eyes which their prominence produces gives rise to proportional troubles.

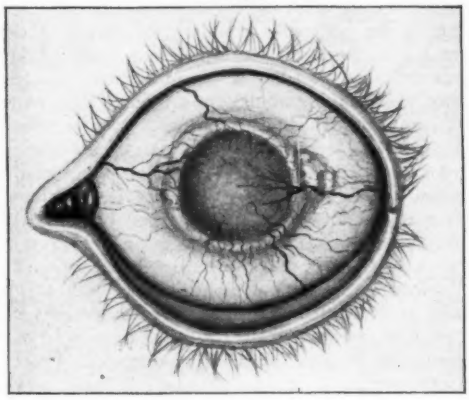
The immediate cause of the proptosis has been attributed to vascular engorgement of the orbit, to contraction of the unstriped muscle tissues in the orbit due to undue stimulation by the sympathetic nerve, to increase of intra-orbital fat, which is unlikely, and to increase in size of the ocular muscles with edema and inflammation.

The following signs should be looked for: deficiency of convergence; retraction of the upper eyelid; infrequency and incompleteness of the blinking reflex and ocular palsies which may develop in Graves' disease; ophthalmologia interna, however, does not occur.

Exophthalmos after thyroidectomy—Exophthal-







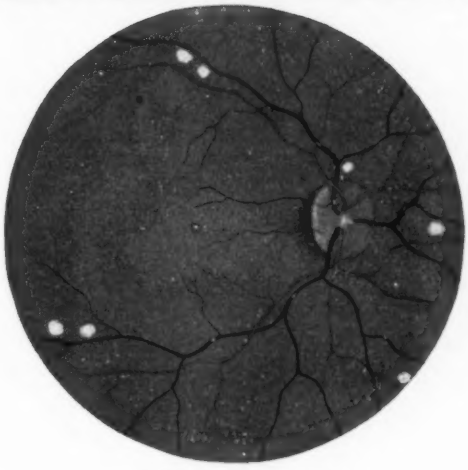


FIGURE 12 *(top, left)*. Albinotic fundus. Note the choroidal vessels which are visible except in the macula where the retinal pigment remains.

FIGURE 13 *(top, right)*. Diabetic "snowflake" cataract. Note the punctate grayish white opacities affecting the anterior and posterior cortices of the lens.

FIGURE 14 *(center, left)*. Sclerokeratitis in tuberculosis. Note the diffusely gray appearance of the cornea and the bluish gray elevation of the sclera surrounding the cornea.

FIGURE 15 *(center, right)*. Miliary tuberculosis of the eye. Note scattered grayish white, rounded tubercles.

FIGURE 16 *(bottom, left)*. Conjunctival chancre.

FIGURE 17 *(bottom, right)*. Disseminated choroiditis in a syphilitic adult patient.

mos may increase after thyroidectomy in certain cases even though other symptoms may diminish. This has been attributed to increased secretion of the thyrotropic hormone by the anterior pituitary gland.

Treatment is the same as that of the primary disease. Prevention of involvement of the cornea may require closing the eyelids by temporary or permanent adhesions or even orbital decompression.

The ophthalmologist's interest in treatment is concerned mainly with the differential diagnosis of thyrotoxic and thyrotropic exophthalmos. The importance of a correct differentiation is clear when one considers the disastrous results produced by treating the thyrotropic type by thyroidectomy. Early diagnosis, which is always more difficult, should be our goal and it is well to remember that all cases of this type may not be as clearcut as Mulvany[21] suggests. Mann[22] points this out. She believes that clinically in most cases with a raised basal metabolic rate (thyroxin excess) not due to a thyroid adenoma, there is a pituitary element manifesting itself by lid edema and limitation of some of the ocular movements. Her classification therefore includes a third group of cases which manifests signs of excess thyroxin occurring simultaneously with signs of excess of thyrotropic hormone.

Treatment of thyrotropic cases is difficult as they cannot be given thyroid extract and must be handled more or less symptomatically. It is worth mentioning that in spite of difficulties, all of Mann's thyrotropic cases did well. When proptosis is increasing, tarsorrhaphy is worthy of trial before undertaking a formidable procedure like the Naffzinger operation of orbital decompression. For more details of treatment see Williams[23] and Means.[24]

Hypothyroidism—A lowered basal metabolic rate may lead to a lower content of thyroxin in the cells. The administration of thyroid extracts usually benefits myxedema and eye symptoms and signs. Insufficiency of accommodation and convergence, dryness of the conjunctiva and thinning of the eyelashes and eyebrows are some common signs of hypothyroidism. Incidence of cortical cataract in hypothyroidism is high according to Lemoine.[25]

DISEASES OF THE BLOOD

Malaria—It is well known that dendritic corneal ulcers are usually a sequel to a febrile attack and they seem to be especially common in malaria; thus, of 64 cases of corneal pathology in malaria, 60 were of this nature. In the malignant form of this disease, retinal hemorrhages may occur, especially when marked anemia is present. A case in which the central retinal artery was blocked by parasites has been recorded.

INFECTIONS AND INFECTIOUS DISEASES

Rheumatism-arthritis—In rheumatoid arthritis any eye tissue may become inflamed and secondarily degenerated. Iridocyclitis and uveitis are fairly common serious complications.

Nose and throat diseases—Optic neuritis, retrobulbar neuritis, iritis and uveitis, keratitis and disturbances of motor function (paralyses, spasm) are fairly common complications of diseases of the accessory nasal sinuses.

Tuberculosis—Ocular tuberculosis is rarely encountered in patients with active tuberculosis but more commonly among patients with healed lesions. Virulence of the organism and the immune state of the patient account for the nature of the eye lesion. Some of the eye lesions observed are periostitis, dacryo-adenitis, conjunctivitis (tubercle usually; coxcomb granulations are rare). Phlyctenular conjunctivitis, scleritis (usually sclerokeratitis) (Figure 14), interstitial keratitis, uveitis (iritis, iridocyclitis and choroiditis), retinal periphlebitis, retinal tubercle (Figure 15), recurrent vitreous hemorrhages and optic neuritis may be found in tuberculosis. General treatment is most important (newer antibiotics seem to be beneficial, especially dihydrostreptomycin). Desensitization with tuberculin is beneficial according to some authors although in my opinion the treatment may be largely nonspecific in its effect and the diagnosis is often erroneous.[26]

Brucellosis—The ocular involvements vary within wide limits, some of which may not truly represent ocular signs of brucellosis. Brucellosis may be the cause of endogenous uveitis in 5 to 10 per cent of cases according to Woods.[27] It is a suspected cause in only 3 per cent of patients seen by me in New York City. In some cases reported in which the ocular diagnosis was reasonably certain, the ocular lesions were serious and resulted in severe impairment or complete loss of vision. Green[28] reported 4 cases. The ocular findings were keratitis and corneal ulceration, retinal edema with hemorrhages and subjective complaint of metamorphopsia, optic neuritis and central chorioretinitis. Optic atrophy has been recorded. Papilledema occurred in 3 of 63 patients who suffered from brucellosis.

Harris[29] described chronic brucellosis with recurring iritis, hemiplegia and death from a complicating diffuse encephalomalacia. Walsh[30] observed 2 cases which had been treated for syphilis for several years, which were later established beyond reasonable doubt to have been based upon false positive Wassermann reactions. I treated one patient for rapid ocular fatigue, the cause of which was finally determined to be brucellosis by Dr. Hyman.

Syphilis. Primary stage—Chancre of any part of the conjunctiva (Figure 16) or eyelid associated with enlargement of preauricular lymph nodes.

Secondary stage—Paralysis or paresis of the extra-ocular and intra-ocular muscles, mucous patches of the conjunctiva, conjunctivitis, scleritis, iritis and syphilitic chorioretinitis (Figure 17).

Tertiary stage—Various oculoneurologic disturbances, chronic orbital periostitis and gumma of the orbit, Argyll-Robertson pupils (react to accommodation but not to light), keratitis, uveitis, and atrophy of the optic nerve.

Tabes dorsalis—Tabes does not seem to produce characteristic ocular symptoms or signs except perhaps optic atrophy. Ocular signs are common but they are usually due to the coexisting chronic syphilitic meningitis. Because the association is so frequent, there is a tendency to consider them as symptoms of tabes, but they sometimes occur in syphilitic cases without sclerosis of the posterior columns.

General paresis—The ocular symptoms observed are similar to those noted in tabes and chronic syphilitic meningitis. Unequal pupils are a particularly early symptom in paresis (Figure 18).

Congenital syphilis—The most common lesions are keratitis, iritis and optic neuritis. When congenital syphilis exists the eyes are affected in 75 per cent of the cases.[31] The Wassermann reaction was found to be positive in 53.8 per cent of cases of interstitial keratitis.[32] Interstitial keratitis (Figure 19) usually occurs between the ages of 5 and 20 years but may develop later. However, this form of keratitis is rarely acquired. In treating interstitial keratitis, first dilate the pupil with atropine and then administer antisyphilitic treatment which may shorten the course of the disease which usually persists from three to eight weeks.

Gonococcal infections—Gonococcal blood infection with arthritis from ophthalmia neonatorum is rarely seen.

Iritis with associated cyclitis occurs in some cases of gonorrheal arthritis, usually subsequent to involvement of the joints; it is usually bilateral, and the first attack is at times characterized by the presence of a yellow translucent exudate in the anterior chamber. Recurrences of iritis are frequent, but are not associated with the marked constitutional disturbances which are present in a first attack.

ALLERGY—OCULAR MANIFESTATIONS

Types of allergy—Clinical allergy does not always meet the criteria of demonstrable circulating antibodies according to the hypothesis of Von Pirquet founded upon the investigations of Arthus, Ehrlich and his own work.

Because of Duke and others a concept based upon the gradual development of an increasingly more elaborate body mechanism of resistance to changes in the external and internal environment through phylogeny, allergy (humoral, bacterial, physical) may be considered as the result of hyperfunction of the body's defense mechanism ("alarm reaction") of Selye. However, these types are not mutually exclusive.

Physical allergy—In the physical or reflex type of allergy vasospasm and anoxia cause increased capillary permeability, liberation of histamine and vascular injury. The allergic wheal is typical of this reaction.

Bacterial or tissue allergy—Cell injury results from an antigen-antibody type of reaction coupled with the primitive vascular reaction. No circulating antibodies are found in the blood stream. Granulomatous inflammation may be considered the normal physiologic prototype of this type of hyperfunction of the resistance mechanism.

Humoral allergy—Circulating antibodies (probably from lymphocytes) are present in the blood serum in addition to the primitive vascular reaction and antigen-antibody reaction in the cells which are retained. The result of this hyperfunction is suppurative inflammation.

The specific treatment of ocular allergy depends upon the etiology. Special attention must be given to the history, effects of seasons, exposures, inhalants in the home, and to the occupational and recreational environments. Skin reactions are important and may indicate ocular sensitizations. Frequently they are negative when conjunctival reactions are positive,[33] and in some the condition of the conjunctiva or cornea is such that local eye tests are contraindicated. Determination of pollen sensitization and desensitization should be carried out as in hay fever and asthma. Optimum doses repeated for many weeks or months may be necessary if relief is to be obtained.

Perennial symptoms necessitate the consideration of all animal emanations, dust, cosmetics and miscellaneous allergens, including the possibility of fungi and occupational dusts which occur in the patient's environment. Desensitization with antigens containing all important ones may be necessary, carried out as outlined for pollens. Skin tests, history and therapeutic results must determine the allergens necessary for therapy. Environmental control affords adequate protection. Food allergy needs serious study in the perennial cases. It may be a complicating factor as in seasonal hay fever. Elimination or test negative diets must be used for diagnosis and control. Pulse diet[34] studies are often helpful.

Allergy from the ingestion and local application of drugs must be kept in mind. Bacterial allergy is sometimes controlled by treatment with minute doses of vaccine,[35] by nonspecific therapy and by surgery for improving drainage or by eliminating foci. In tuberculosis of the eye, careful desensitiza-

tion with tuberculin may be beneficial. Woods successfully used a slow and careful desensitization with Davy's bouillon filtrate in ocular allergy to tuberculin.

Preoperative desensitization to lens antigen[36] before cataract extraction is sometimes of value. In sympathetic ophthalmitis desensitization with uveal pigment seems to be helpful at times.

The antihistamines by mouth (THEPHORIN, BENADRYL, PYRIBENZAMINE) and locally (ANTISTINE and PRIVINE) are often of value in relieving annoying symptoms.

OUTLINE OF ALLERGIC AFFECTIONS

Head—Migraine headache.

Eyelids—Angioneurotic edema, eczema, blepharoconjunctivitis, blepharitis sicca and ulcerosa, and contact dermatitis (from powder, soap, drugs and plants).

Conjunctiva—Conjunctivitis associated with tearing and photophobia and especially with itching (cosmetics, foods, drugs, especially atropine and penicillin), bacteria, pollen and animal emanations; blepharoconjunctivitis, vernal catarrhal conjunctivitis (associated with eosinophilia and with itching).

Cornea—Phlyctenular keratitis, syphilitic and tuberculous interstitial keratitis.

Iris—Iritis and iridocyclitis (tuberculosis, syphilis, chronic infection), possibly sympathetic ophthalmitis (allergy to uveal pigment).

Lens—Cataract; phaco-anaphylactic or associated with atopic eczema.

Retina—Possible detachment of the retina.

Optic nerve—Papillitis.

VITAMIN DEFICIENCY AND OCULAR PATHOLOGY

Vitamin A deficiency may be suggested by night blindness, hypofunction of the meibomian glands, desiccation of the conjunctiva or cornea, blepharitis, meibomianitis, hordeolum, episcleritis, ulcers of the cornea and keratitis profunda.

Vitamin D deficiency may possibly be indicated by keratoconus.

Vitamin B deficiency (especially of two of its components (thiamine and riboflavin) may be mild or severe. A mild thiamine deficiency may be suggested by poor vision, dryness and burning sensation of the eyes, and conjunctivitis. Marked deficiency may be manifested by toxic amblyopia, chorioretinitis, and retrobulbar neuritis. Hyporiboflavinosis may be suggested by circumcorneal injection, with invasion of the cornea by capillaries, rosacea keratitis, itching, burning, a sensation of roughness of the eyelids, lacrimation, photophobia and twilight blindness. This condition may be confirmed by noting the associated oral and dermal lesions—encrusted moist fissures at the angles of the mouth, glossitis, desquamation of the skin, and seborrheic accumulations in the nasolabial folds, on the alae nasi and in the nasal and aural vestibules.

Vitamin C deficiency may be indicated by increased capillary fragility and a tendency to hemorrhage into the vitreous or in the retina, especially in persons who have arteriosclerosis or diabetes.

Vitamin K deficiency. Because vitamin K assists in the normal coagulability of the blood, lack of this vitamin may be suggested by the presence of hemorrhagic retinitis.

Vitamin P deficiency is associated with increased capillary fragility in diabetic retinopathy in some patients.

CERTAIN EYE DISEASES WITH GENERAL CAUSES

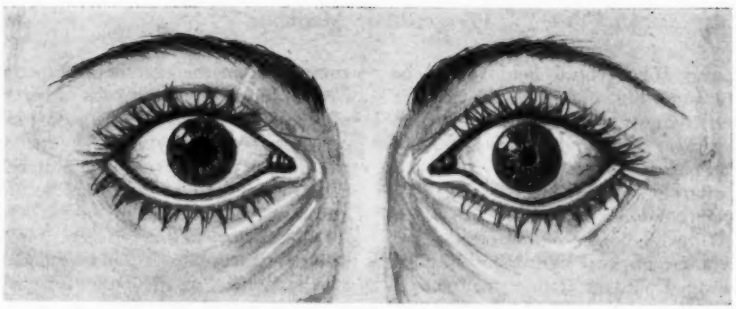
THE ability to diagnose glaucoma is important to every physician, for failure in diagnosis too often results in unnecessary blindness. Acute glaucoma may be mistaken for conjunctivitis, keratitis or iritis and may result in total blindness unless the proper diagnosis is made. The increased pressure of the eyeball is usually so marked that palpation will permit diagnosis without the use of a tenometer. However, it would be desirable if every physician had a hypertension indicator[1] to aid in this important differential diagnosis.

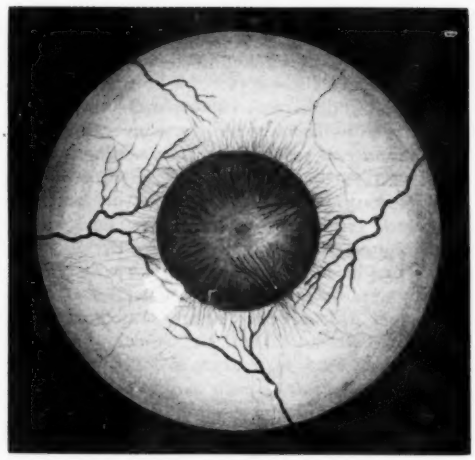
Glaucoma secondary to uveitis—If inflammation of the uveal tract persists, glaucoma will often develop. In addition to local treatment and often surgery to lower tension, general causes (tuberculosis, syphilis, brucellosis and chronic "focal" infections) should be sought and treated.

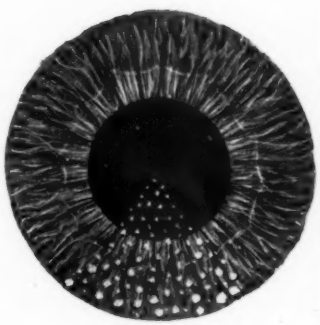
Chronic primary glaucoma—Chronic primary noncongestive or simple glaucoma is insidious in onset and unfortunately is often the cause of total blindness. Although it is usually seen after middle life, it may occur in young people—for example, a boy 19 years of age was operated upon by the writer. In the early stages of the disease even a well-trained ophthalmologist may fail to make the diagnosis. Rapid changes in refraction, necessitating frequent prescribing of stronger reading lenses, constitute one of the earlier symptoms of the disease. Many patients consult refractionists who sometimes prescribe several pairs of glasses until their visual fields and even central vision are affected.

A history of colored rings around lights, rapidly changing refraction, radiating pains above one or both eyes which may awaken the patient at night and beginning contraction of the nasal fields with small test objects, are fairly early important signs. Since the intra-ocular pressure may not be continuously elevated, it should be taken repeatedly before the diagnosis is confirmed or discarded.

The influence of dark adaptation and mydriatics which usually cause a rise in pressure in glaucomatous eyes are justifiable means for detecting







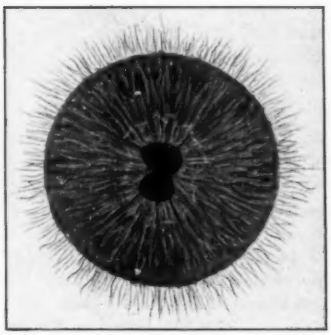


FIGURE 18 *(top)*. General paresis. Note the inequality of the pupils.

FIGURE 19 *(center, left)*. Interstitial keratitis. Note the central gray opacity in the stroma of the cornea and the deeply placed brushes of blood vessels extending into the cornea.

FIGURE 20 *(center, right)*. Optic nerve in glaucoma. Note the deep cup and the way the blood vessels bend over the edges of the depression. *(insert)* Cross section of the optic nerve showing advanced glaucomatous cupping.

FIGURE 21. Appearance of the cornea and iris in iridocyclitis (uveitis). *(bottom, left)* Note the keratic precipitates (K.P.) deposited on the posterior surface of the cornea in the form of a triangle. *(bottom, center)* Acute iritis with posterior synechiae. Note the irregularity of the pupil caused by posterior adhesions to the lens capsule.

FIGURE 22 *(bottom, right)*. Immature senile cataract. Note the grayish white, somewhat triangular opacities extending from the lens periphery toward the pupil.

incipient glaucoma. The central field should be studied with minute test objects to detect the earliest perimetric signs of incipient glaucoma. These are enlargement of the blind spots and small blind areas called paracentral scotomas in the visual field near the point of fixation.

The typical glaucomatous cup (Figure 20) which extends to the temporal border of the optic nerve is unfortunately a late physical sign. Many cases of blindness resulting from the failure of general physicians to diagnose glaucoma have been reported and 2 patients were seen by me within two weeks who lost their vision because the diagnosis of glaucoma was not made. In one case a general practitioner diagnosed conjunctivitis instead of glaucoma and in the other the general physician considered the condition to be that of facial neuralgia when the diagnosis should have been glaucoma following cataract extraction. As early as 1924 Lowell[37] reported 7 cases in which there was failure on the part of physicians and refractionists to diagnose glaucoma, resulting in great loss of vision.

Although chronic primary glaucoma is commonly considered to have no general cause, a most careful physical and laboratory examination is indicated and several possible contributory factors are usually found. Psychologic factors, chronic infections (chronic sinusitis[38]) and allergies[39] should receive special consideration. Physicians should be careful in the diagnosis of conjunctivitis, iritis and facial neuralgia because glaucoma may be the true diagnosis and blindness may result. A hypertension indicator is available to study eyeball tension.

Optic atrophy—Syphilis and brain tumor are common causes and frontal lobe tumors may produce atrophy on one side and papilledema on the other (Foster Kennedy syndrome).

KERATITIS AND GENERAL CAUSES

Phlyctenular keratitis—Note a small pinkish elevation, the apex of which ulcerates and then heals without scarring, in a localized congested area at the limbus. Phlyctenules may also occur on the bulbar and palpebral conjunctivas and usually disappear in from 8 to 10 days, but may recur over periods of months or years. The cornea may be involved directly or by extension from the conjunctiva (phlyctenular keratitis). This type of ulcer, which may be progressive, may be followed by a leash of superficial blood vessels which forms a band in the furrow created by the ulcer (fascicular keratitis[40]).

The disease occurs frequently in debilitated children and in those who suffer from the tuberculous or the "so-called" scrofulous diathesis. It is especially frequent among the so-called "lower classes," in whom inadequate diet and unsanitary conditions in general are contributory factors; also in children debilitated from disease and in those recovering from the exanthems, especially measles.

One frequently sees such other manifestations of the predisposing diathesis, as swelling of the cervical lymphatic glands, adenoids, eczema, rhinitis, blepharitis, chronic otorrhea, etc. Sometimes, however, the affection occurs in children apparently in good health.

Iridocyclitis. Causes—Except for cases due to trauma, iridocyclitis is a local manifestation of disease elsewhere in the body. Twenty per cent of cases are caused by syphilis, 17 per cent by tuberculosis, 29 per cent by infection of the tonsils, teeth and paranasal sinuses, 9 per cent by other less common foci of infection, 3 per cent by gonococcal infection, 10 per cent by combined infections, 3 per cent by brucellosis and 9 per cent by undetermined causes. Gout and diabetes are responsible for a relatively small number of cases. In acute iridocyclitis the importance of tuberculosis as an etiologic factor is relatively small, while that of gonococcal and focal infection is great. In chronic iridocyclitis, tuberculosis plays an important part, while cases due to syphilis and gonorrhea are much less common. While the term "rheumatic iridocyclitis" was formerly employed it is now recognized that rheumatism and iridocyclitis are both caused in many cases by chronic "focal" infection. Certain cases occur during an acute respiratory infection or during acute infectious diseases such as influenza, measles or scarlet fever and are apparently caused by the agents responsible for these diseases.

Other diseases causing uveal inflammation are Boeck's sarcoid and the chrome form of brucellosis. Every effort should be made to determine the etiology of iridocyclitis but in many cases the cause remains unknown. The clinician is often forced to guess the etiology. That the pathologic and clinical diagnoses often do not agree is clearly shown by our studies.[26]

Acute iridocyclitis—In acute diffuse iridocyclitis due to various causes, deep congestion, lacrimation and pain are manifested. The pupil is small, aqueous, cloudy, and the iris markings obscured by swelling and a fibrinous exudate. Congested iris vessels are visible. Vision is only slightly affected in the early stages by the cloudy media and by spasm of the ciliary muscle which may cause one or more diopters of myopia. The normal connection between the posterior and anterior chambers becomes blocked with seclusion of the pupil, causing fluid to collect in the posterior chamber. The iris bulges forward at the periphery (iris bombée), and becomes adherent to the cornea. Secondary glaucoma may result with loss of vision. When exudation from the ciliary body is severe, dense cyclitic membranes form behind the lens resulting in retinal detachment or phthisis bulbi by virtue of subsequent contraction.

Chronic iridocyclitis—In a number of cases, the inflammation begins insidiously and continues without showing signs of acute inflammation. Symptoms of chronic iridocyclitis differ from acute iridocyclitis in that pain and congestion are much less marked. Large keratic precipitates (K.P.) are seen on the corneal endothelium; in older cases they contain granules of pigment and are arranged in the form of an irregular triangle, with the apex upward (Figure 21A). Posterior synechias are usually numerous (Figure 21B); complete occlusion of the pupil with iris bombée and secondary glaucoma is not infrequent. Occlusion of the pupil and cataract due to chronic inflammatory changes are seen more in chronic than acute iridocyclitis.

Cataract—(See Diabetes).

Complicated cataract—Due to intra-ocular pathology. Principal causes are chronic uveitis, retinitis pigmentosa, and retinal detachment. In these conditions the normal nutrition of the lens is disturbed and a characteristic type of opacity develops. In high myopia with fluid vitreous, cataract is common, but is usually of the perinuclear variety. Typical complicated cataract begins in the cortex near the posterior pole of the lens, becoming larger and thicker as new fibers are laid down. Later more of the cortex is affected and the entire lens may become opaque.

Cataract due to systemic disorders—In tetany following operations on the thyroid, a rapidly progressive type of cataract sometimes develops. It is usually bilateral of the cortical type, and may progress so rapidly that removal of the lens is required in from two to six months. If seen very early, the use of parathyroid hormone and calcium may limit the extent of the opacity. In older persons with mild diabetes, the cataract is usually of the senile type. The use of dinitrophenol for reduction of weight has caused a rapidly progressive type of cataract. In myotonic dystrophy a peculiar type of cataract occurs. Opacities begin beneath the lens capsule and around the posterior suture lines, and are seen as fine white flakes, some of which give a play of color in focal light. The condition progresses over a period of several years to the picture of mature cortical cataract. In generalized dermatitis, a similar type of cataract develops known as cataracta neurodermatica.

Irradiation cataract—The use of radium and roentgen rays in large doses has frequently given rise to a cataract of a peculiar type. Opacities begin in the posterior cortex until the entire lens becomes opaque. Progress is slow and opacification of the lens may be incomplete.

Senile cataract (Figure 22)—This includes any form of spontaneous opacification of the lens occurring in persons past middle age without other evidence of ocular disease which would account for it. The exact etiology is unknown so the best treatment is to improve local ocular nutrition and general health. Removal of foci of chronic infection should be considered.

Ptosis—Inability to raise the upper eyelid is named paralytic ptosis when a branch of the oculomotor pair of nerves which innervates the levator of the upper eyelid is affected. Ptosis caused by myositis of the levator of the eyelid is rare. The nerves may be affected in the orbit, in their course, at the base of the skull or in the region of the nuclei. Isolated ptosis is often of traumatic origin (fracture of the orbit) but more commonly syphilitic (orbital periostitis, basal gummatous meningitis or tabes).

Sinusitis (frontal, sphenoid) and tumors of the orbit are less common causes.

Ptosis in complete paralysis of the oculomotor nerve is accompanied by turning of the eyeball downward and outward by the normally functioning fourth (superior oblique) and sixth (lateral rectus) nerves, a dilated pupil which does not react to convergence and paralysis of accommodation. The site of these lesions is usually basal, bulbar or orbital.

Ptosis may be associated with other cerebral or general symptoms (botulism, diphtheria and in Weber's syndrome). In Weber's syndrome, hemiplegia is contralateral to the ptosis and a lesion in the region of the peduncle is usually causal (tumor, hemorrhage, syphilis or tuberculosis). A temporosphenoidal abscess (otitis, sinusitis) may be causal. Prognosis and medical treatment depend upon the cause of the ptosis. Surgery is resorted to when medical treatment fails.

SUMMARY AND CONCLUSIONS

It is evident from this brief outline of some of the symptoms, physical signs and diseases occurring in the practice of the ophthalmologists that he requires the cooperation of general physicians and specialists to a high degree if he hopes to prevent blindness and cure the conditions he sees.

It is equally evident that knowledge of certain facts presented here is of great importance in the diagnosis and treatment of many conditions which are seen by physicians and specialists. It is obvious that the best interests of the public and the public health will be served if each specialist recognizes the importance of other specialties and the unusual qualifications of certain specialists and cooperates by seeing that treatment is administered by the man most skilled in treating the condition under observation. There is no longer justification for disregarding modern aids in diagnosis. With an ophthalmoscope, the ordinary flashlight and the use of certain instruments, for example the hypertension indicator, we can greatly improve our accuracy and effectiveness in the diagnosis and treatment of our patients and prevent much blindness.

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LADY OF SHALOTT

One of a series of "anatomic" drawings by Pavel Tschelitchew.

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The Pathology and Present Treatment of Ovarian Cysts

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Obstetrics has recently passed through a period of critical survey. Now gynecology is in the spotlight of inquiry.[31,32] One must admit that much censure has been justified because of traditional radical surgery involving ovarian cysts. Cystic tumors of the ovary are the most common pelvic neoplasms,[34] and it is logical that they should be often considered serious lesions, worthy of prompt excision. However, most gynecologists believe that too much unjustified surgery is done on the ovary today. We hold that the worst offenses are (1) the removal of the entire ovary when only a small physiologic cyst is present, (2) the removal of the ovary because of a moderately large, benign cyst which could have been resected,[16] (3) the removal of both ovaries, either for surgical convenience[4] or, "to avoid cancer at some later date."

Relief of the patient's troublesome signs and symptoms, with conservation of ovarian tissue, should be the physician's prime objective—unless malignancy or pelvic cellulitis makes ovariectomy mandatory. In line with this tenet, we believe that a better understanding of ovarian pathology, more careful preoperative diagnosis and a keen surgical conscience will insure the patient's best interests. It seems proper, then, to consider (1) certain functions of the normal and abnormal ovary, (2) the physiologic ovarian cysts and their signs and symptoms, (3) the pathologic ovarian cysts—with special reference to those which are definitely cancerous or have malignant potentials and (4) the modern medical and surgical treatment of all adnexal cysts.

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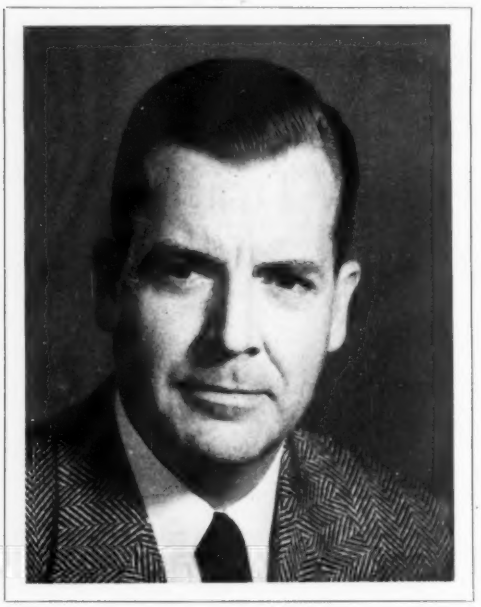
FUNCTIONS OF THE NORMAL AND DEFICIENT OVARY

The fully-functioning ovary performs at least seven duties.[6] These range from the production of fertilizable ova to the formation of two hormones vital to the health and welfare of the woman. However, these functions are not always present in an individual gonad or even in both ovaries taken together. Some ovarian capacity may be absent during a part or most of the woman's functional life. It is usually assumed that ovulation and hormone production are equally divided between the two ovaries. This, unfortunately, is not always the case. The justification for the removal of one ovary just because the woman has another is therefore fallacious. Bonney states that this is "like the abstraction of one of two shillings from another person's pocket—the one left behind may be no good."[5]

The removal of one ovary is justified by some on the grounds that one ovary will do the work of two. This is likened to the excision of one testicle, one lung or one kidney, with the assumption that adequate function will continue if the burden is not too great. This disregards the physiologic function of the ovary. Of all the endocrine glands, the ovary is the only one which does not maintain the same physiologic unity continuously. Not only physiologic changes involving the cyclic production of the hormones, but also a variation in the physical forces within the ovary occur periodically. Great stress is caused within the ovary by the persistent rise and decline of follicles. One ovary may not be adapted to the pace of a menstrual



RALPH C. BENSON

cycle which generally seems to require two.[17]

After the removal of one ovary in experimental animals, the remaining gonad increases in size and produces a greater number of ova. If it remains healthy, it will adapt itself to this demand. However, it often seems to "wear out more rapidly" and an earlier climacteric appears to result.[14] Beyond a certain point, the removal of ovarian tissue results in disturbed function. If less than one-fourth of an ovary is left, complete failure of ovulation and other signs of ovarian failure are said to result.[49] Some of these data from animal experimentation are applicable to the human. It is possible, therefore, that the removal of a single ovary, pathologic though it may be, can eliminate all truly active ovarian tissue.

We hear physicians casually mention the removal of a "little cyst on the ovary." Such a cyst is usually inconsequential, but unfortunately it may often be the reason for the removal of the entire ovary, found beneath the cecum at the time of an interval appendectomy. Women can be forever grateful to McBurney because the left ovary cannot be removed easily through his incision.[19] Similarly, general surgeons have been accused of holding the left ovary in greater esteem than the right, since the left is the one usually allowed to remain.[31] Such surgery is almost always meddlesome and unnecessary. Save for extensive endometriosis, chronic tubo-ovarian abscess formation or malignant disease, conservation of both ovaries, or a portion of both, is almost always possible.[5]

PHYSIOLOGIC OVARIAN CYSTS

It is unfortunate that until recently the treatment of *all* cystic changes in the ovary has usually been surgical and radical. Fortunately, however, within recent years increasing conservatism has characterized pelvic surgery. This trend has been prompted by the realization that many of the smaller cysts, for which the ovaries were sacrificed, were actually normal transient structures. Moreover, it is now appreciated that certain ovarian cysts are the result rather than the cause of hormone aberration. Finally, we have come to realize that there is a medical approach to the resolution of the nonproliferative ovarian cysts. Specifically, it has been demonstrated that the administration of estrogens or androgens often causes or aids in the resolution of physiologic ovarian cysts with subsequent resumption of normal ovarian function.[23,26]

An ovarian cyst, by definition, is a sac or space filled with a liquid or semisolid material. This interpretation is sometimes provided belatedly by the pathologist in his description of microscopic ovarian defects which may be without clinical significance. Some cysts, however, are either strikingly symptomatic or are so large, tense and persistent as to be of probable gynecologic importance.

So far as mass formation is concerned, it is vital for one to decide what constitutes a clinically important cyst. Miller[32] states that persistent enlargement of the ovary beyond 5 cm. generally signifies true pathology. But even this may be within physiologic limits. Cook[8] has noted that in most cases of simple cystic tumefaction enlargement disappears within five months without specific therapy.

ETIOLOGY OF OVARIAN CYSTS

The cause of cystic ovaries may lie within the ovary itself or may be due to functional pathology outside the pelvis.[25] Geist[15] states that the development of follicular cysts was thought by Pfannenstiel and A. Martin to result from chronic oophoritis with fibrosis of the capsule.

TABLE 1
OVARIAN CYSTS

NON-NEOPLASTIC CYSTS	NEOPLASTIC CYSTS	SECONDARY CYSTIC DEGENERATION IN
(Parovarian cysts)	† Cystadenoma	‡ Granulosa-theca cell tumor
Germinal inclusion cysts	Papillary serous	Dysgerminoma
‡ † Follicular cysts	Papillary pseudomucinous	Mesonephroma, etc.
‡ † Lutein cysts	† Cystadenocarcinoma	
Corpus luteum cysts	Papillary serous	
Theca lutein cysts	Papillary pseudomucinous	
Corpus luteum hematoma	† Cyst teratoma (dermoid)	
† Endometrial cysts	Teratoma	

† Common or important
‡ Functional

Mechanical hindrance to the rupture of the follicle resulted, with secondary vascular disturbance following. Gebhard and Orth considered follicular cysts to be normal physiologic variations. Polycystic ovarian disease is thought by Weed, et al.[50] to be produced by follicular atresia resulting from persistent vascular congestion. They further postulate that follicular atresia is the result of faulty nutrition to the ova.

Other investigators suggest that failure of ovulation and/or excessive production of follicle-stimulating hormones is a cause of follicular cysts.[34] The injection of sizable amounts of gonadotropin is also known to produce lutein cysts.[51] Jacobson[21] has pointed out that the "ovarian white line," or point of condensation of the peritoneum and tunica albuginea, is often dense and unyielding. Hence, small follicles appearing at or beneath this line do not rupture and thereby cause circulatory impediment. Further scarring at the white line then occurs, and this aggravates the situation. Kurzrok[25] has stated that excessive use of estrogen by the endometrium, as in endometrial hyperplasia, may require the ovary to develop more follicles for the production of more estrogen, and this leads to a vicious cycle. However, there are numerous other cystic ovarian tumors which have no known cause. A simplified classification of ovarian cysts is shown in Table 1.

A brief review of certain of these cystic structures may aid in emphasizing their significant features:

Parovarian cysts are not true ovarian cysts, but are remnants of the wolffian system and may enlarge to about 10 cm. in diameter. They are nonfunctional unilocular cysts and cause signs and symptoms by pressure only. They almost never become malignant.

Germinal inclusion cysts are insignificant in size and are seen just beneath the surface of the senile ovary. They are formed by an infolding of the surface of the germinal epithelium and have no proved clinical significance. Cystomata theoretically may develop from these cysts.

Follicular cysts are also called retention or simple cysts. They are usually small (1 to 6 cm. in diameter) and are not proliferative. There is still some debate as to whether they are actually functional, but they never become cancerous.

During a woman's functional years the rupture of a graafian follicle is a normal, almost monthly phenomenon. If ovulation, with the usual extrusion of the ovum, fails to occur, spontaneous regression or atresia of the follicle usually results. However, a persistence of the follicle, despite degeneration of the ovum, may occur. Clinically, such cyst formation is common at puberty or in the premenopause. These cysts are frequently noted in prolapsed ovaries or in chronic pelvic inflammatory disease.

An atretic follicle is not functional so far as effective hormone production is concerned despite the residual living theca lutein cells. Nevertheless, a persistent follicle or group of follicles which follow repeated failures of ovulation, perioophoritis, or overstimulation of the ovary by the anterior pituitary, may result in hyperestrinism and dysfunctional uterine bleeding. Arrested proliferation of the endometrium or endometrial hyperplasia is the expected uterine reaction when functional granulosa cells are present in such ovaries. Both ovaries are noted to be microcystic or, if the individual cysts are sizable, a polycystic ovary is described.

Occasionally, a functional solitary follicular cyst is discovered. This is always a retention cyst and is not a true neoplasm. It may reach a size of 6 cm. in diameter. The difference between such a tumor and a polycystic ovary is

quantitative rather than qualitative. The cyst walls in either case are thick and opaque. The tumor surface is smooth and a serous amber fluid is contained within the lumen. This liquid, high in estrogen, is both a secretion and a transudate. Because of the increased intracystic pressure and defects in the cyst walls, numerous cysts coalesce to form larger cysts. Compressed theca lutein cells line the cysts. Considerable difficulty is often met in distinguishing these cells because of age of the cyst, fluid pressure and infection. Often only a few islands of recognizable cells remain. Large cysts compress the remainder of the ovary and destroy much of the ovarian tissue save that at the hilus. Pain, due to torsion, or dyspareunia, may occur. Hemorrhage into the cyst, with rupture and intraperitoneal bleeding, may constitute a surgical emergency.

Follicular cysts are probably not the cause but the result of hormone imbalance. This depends, we think, upon (1) the failure of ovulation and resulting unopposed estrogen and (2) uncompensated anterior pituitary activity.

Lutein cysts may be granulosa lutein cysts (as in prolonged, active corpora lutea), or theca lutein cysts (as in association with hydatidiform mole or chorionepithelioma).

Corpus luteum cysts are not uncommon. They are in every sense functional, non-neoplastic tumors of the ovary. The life cycle of the corpus luteum is ordinarily well regulated, from ovulation to about three days prior to the onset of menstruation, when involution occurs. Generally, the normal regression of a corpus luteum requires organization of the blood, which ordinarily fills the center of the corpus luteum after extrusion of the ovum. However, cyst formation by excessive blood and serum within a corpus luteum just before the vascularization stage may interfere with the normal regression of the lutein layer. It is more than a hemorrhagic corpus luteum. Discomfort, delayed menstruation and menorrhagia result.

In pregnancy, the corpus luteum is naturally large (4 to 6 cm. in diameter) and is usually cystic until after the seventh month of gestation. Occasional rupture of these cysts in the nonpregnant or pregnant state results in intraperitoneal bleeding. Laparotomy is required if this bleeding is excessive—and it is sometimes profuse. Occasionally, the clinical history suggests ectopic pregnancy with hematoperitoneum, and tubal gestation must be excluded. In hemorrhagic—or so-called "chocolate"—cysts one must also consider endometriosis.

Endometrial cysts—The origin of ovarian (and peritoneal) implants of endometriosis was stated by Sampson[43] to be retrograde menstruation. Small, viable endometrial fragments, he insisted, may be implanted on vulnerable surfaces. This formulation may explain some cases of endometriosis, but not all. Meyer[30] and Novak[34] favor heteroplasia as a cause of endometriosis. This theory suggests that the ovarian and peritoneal surfaces, which were derived from celomic epithelium, retain the parent tissue's potentiality to develop müllerian tissue—even in adult life. Infection, irritation, hormone imbalance or unknown factors have been suggested as the stimuli for this ectopic endometrial development.

However endometriosis arises, such foci in the ovaries become small areas of active endometrial glands and stroma with local bleeding each period. These points are usually multiple and, when small, look like little bluish cysts surrounded by scar tissue. They are, on section, small blood cysts with hemosiderin, pseudoxanthoma cells, chronic inflammatory elements and fibrosis typifying each zone.

Ovarian endometriosis and cul-de-sac implants are commonly associated. Adhesions soon seal off these areas. Persistent retroversion of the uterus, restriction of the pelvic viscera, shotty nodulation and obliteration of the pouch of Douglas are common signs of endometriosis.

Symptoms of endometriosis are infertility, dyspareunia and acquired dysmenorrhea. Patients are usually women over 30 years of age. The menstrual pain is generally pre- or comenstrual, often of a "grinding" type with discomfort referred to the sacrum and rectum.

Endometrial cysts in the ovary grow slowly but may reach a size of 15+ cm. in diameter. The ovary finally becomes but a shell containing old blood elements. In smaller cysts, one might consider the cyst to be of the granulosal type. But no yellow lining is to be seen in ovarian endometriosis.

No papillary processes are recognized within an endometrial cyst. This would aid in ruling out a cystadenoma complicated by internal hemorrhage. Old blood within a cystic cavity, no matter what the cause, will usually be chocolate colored. Brownish material, for this reason,

is not a reliable single criterion for endometriosis. Associated lesions, scar tissue and the history will permit an accurate diagnosis of endometriosis. Consequently, the presence of chocolate-colored fluid in an ovarian cyst does not necessarily indicate its removal.

PATHOLOGIC (NEOPLASTIC) OVARIAN CYSTS

Papillary cystadenoma and cystadenocarcinoma—Proliferative ovarian cysts are all too common. One out of five persistent ovarian cysts more than 5 cm. in diameter is neoplastic and possibly malignant. These lesions represent one of the most discouraging gynecologic syndromes. Our concern is due to (1) difficulties in diagnosis, (2) insidious progress and (3) unsatisfactory therapy. Proliferative cysts do not incite hormone alteration, but they have grave carcinomatous proclivities. The usual age incidence is 45 to 65 years.

THE origin of cystomata is either an inclusion cyst or, more probably, heteroplasia of the ovarian germinal epithelium. We know that malignant cystadenocarcinomata do not arise de novo. The transition from a benign to a malignant cyst is gradual and may take years. The premalignant period is the span of months or years which may permit the diagnosis and excision of a slowly enlarging cyst before true malignant transition and extension occur.

There are two varieties of cystadenomata: serous and pseudomucinous. The former contains thin fluid and the latter slick, oily, semigelatinous material. The incidence of the benign type is about 1:1. Approximately 50 per cent of the serous and 20 per cent of the pseudomucinous cystadenomata are eventually bilateral—even before they become malignant. Some 50 per cent of the serous variety and 5 per cent of the pseudomucinous cystadenomata become malignant. The problem becomes obviously serious when we realize that 70 per cent of all ovarian cancers are papillary serous cystadenocarcinomata. (Only 2 per cent of all ovarian malignancy is represented by the pseudomucinous variety.)

During the late growth period, papillarity becomes more extensive, solid areas form within the tumor and extension of the lesion through the capsule occurs. After exteriorization of the process, ascites (with included cancer cells) forms, and malignant extension to neighboring viscera is rapid. So it is in the period of apparent quiescence that accurate recognition and adequate surgery will cure the patient. After true malignancy is established only about 20 per cent[33, 36] of the cases can be arrested.

MICROSCOPIC CRITERIA OF TRANSITION FROM BENIGN TO MALIGNANT CYSTADENOMATA

1. Excessive proliferation; stratification of epithelial elements.
2. Intricate pattern; increase in glandular spaces.
3. Anaplasia or de-differentiation. Marked mitosis.
4. Sparse stroma.
5. Invasion of septa and capsule by gland elements. Intralocular cyst formation.

Rarely does the operator have the advantage of frozen or rapid section diagnosis. The treatment of the patient will then depend therefore on the surgeon's training in pathology and the calculated risks involved.

Cyst teratoma or dermoid cyst represents 10 per cent of cystic ovarian tumors. About 25 per cent are bilateral. They occur most generally in the early reproductive life. They may be small to moderately large (2 to 20 cm. in diameter). The cysts are rounded, occasionally lobulated with firm areas frequently near the hilum. The capsule is white, glistening, thick and fibrous. The tumor is characteristically free and forward in the abdomen. The cyst is usually filled with greasy, yellow, sebaceous material, often with hair, bone and teeth. Microscopically a dermoid reveals ectodermal and mesodermal elements: a stratified squamous epithelial lining of a cavity supported by a coarse connective tissue stroma. Typical sebaceous glands, with hair follicles and, more rarely, sudoriferous glands, are found in the firm portion.

When one discovers a dermoid in one ovary, the other should be carefully palpated and/or aspirated with a large-gauge needle. Some operators even incise the other ovary to check for a small dermoid on the apparently normal side. Even with a large dermoid cyst, functional ovarian tissue is usually found near the base. This good tissue should be retained after the excision of the cyst. Dermoids rarely become malignant. A very small number of squamous carcinomas developing in cyst teratomata are recorded.

True teratomata are most unusual and when seen are often tumors of infancy and childhood. Such neoplasms are generally discovered after they have become large or obviously malignant. They are semisolid tumors with a firm capsule which may be perforated by the complex histologic structures within. All of the body layers (ectoderm, mesoderm and entoderm) are represented and, in extreme cases, a grossly imperfect fetus may be discovered. Any and all tissues have been noted within a teratoma. They are frequently very malignant and soon involve the other ovary and entire pelvis. The likelihood of misdiagnosing a teratoma as a dermoid is quite remote.

OVARIAN PAIN

TRUE ovarian pain is not common. When it does occur the mechanism is (1) torsion of the pedicle which causes violent and characteristic pain, (2) incarceration of the cyst in the pelvis disturbing bowel and bladder function, (3) infection or cellulitis (endometriosis) of the adnexa and (4) malignancy and extension which produce ascites, pressure discomfort, enlargement of the legs, etc.

Pain from concealed, tense follicles is also a type of ovarian distress. Such ovaries may be found to be unfortunately suspended or adherent so that circulation is impeded. The pain radiates to the thigh or the sacrum and may even simulate appendicitis. Many patients have submitted to appendectomy, which is later followed by oophorectomy for the pain persisting after the removal of the normal appendix.[21]

Heckel[17] believes that ovarian pain can occur in the absence of any demonstrable ovarian pathology. The nature of the underlying disorder is obscure. Pain may be due, however, to too-resistant ovarian tissue (tunica albuginea). In this case, abnormal pressure with increasing follicles impedes follicular growth and the production of ovarian hormone may be reduced. Thus pain and deficient hormone production may be explained in the same way.

Heckel[17] states that when an ovary is removed because of pain, if ovarian insufficiency is present, pain and ovarian inadequacy will increase. Although it is not fully established that hypoovarianism alone is regularly associated with pain, it has been demonstrated that discomfort associated with a residual ovary or ovarian fragment can be checked by the administration of cyclic estrogen[23] or androgen.[26]

Occasionally, lower abdominal pain, even in the presence of an ovarian cyst, may be due actually to urinary tract infection, sacroiliac abnormality or intestinal pathology, and not to ovarian disease.

HISTORICAL BACKGROUND OF TREATMENT

Ovariotomy was first successfully accomplished by McDowell in 1809.[28] For the next 100 years, this operation enjoyed increasing popularity. The procedure was first used only for large ovarian cysts in the debilitated woman. However, with the advent of anesthetics and finally antiseptics, the removal of ovarian cysts from healthier women became standard practice.[39]

In 1872 Battey[3] started the fad of removing normal or relatively normal ovaries for the correction of neuroses, menstrual difficulties and dysmenorrhea. Hegar[18] and Tait[47] soon joined in this nefarious practice. The revulsion of the profession to the sacrifice of normal ovaries was obvious, and Shroeder[44] in 1884 was the first to advocate partial excision of the ovaries to preserve sex function in cases of small cystic degeneration. Finally, a better understanding of ovarian function and of the physiology of menstruation and psychiatric rationale has helped to check the extirpation of normal ovaries. Nevertheless, it has required three generations of gynecologists to reduce unnecessary ovarian surgery, and its eradication is not yet complete.

A great hullabaloo is raised in some quarters when a pregnancy may be interrupted by curettage or hysterectomy. In certain hospitals, routine consultations are required for visiting attending physicians prior to any dilatation and evacuation on younger women. It seems equally sinful to rob a woman of the chance of future pregnancy and a happy, healthy life by ovariectomy for trivial reasons. Nevertheless, frequent removal of ovaries on little or no indication is countenanced by these same institutions.

Mengert has asserted that true ovarian neoplasms should not be approached surgically unless (1) the cyst is "tennis ball-sized" or larger, (2) its presence has been proved by at least two pelvic examinations and preoperatively, (3) there is an ovarian enlargement causing distinct symptoms.

CLINICAL MANAGEMENT OF OVARIAN CYSTS

It is agreed that, despite the foregoing, the differential diagnosis of a cystic ovary encoun-

tered in a specific patient may be difficult and, prior to laparotomy, is often a matter of possibility and probability. The clinical management of such a cystic ovary will, of course, depend upon careful observations obtained from the history and examination. But even these will not, in many cases, give a sure diagnosis. Adequate yet conservative management will often be based upon likelihood rather than certainty—since a laparotomy cannot be a part of every physical examination.

The clinical clues which point to pathologic trends in ovarian cysts are the following:

1. Persistent or enlarging cyst.
   a. Over 6 cm. in diameter in women under 45 years.
   b. Over 4 cm. in diameter in women over 45 years.
2. Continued or severe abdominal pain.
3. Bilaterality of cystic process.
4. Nodulation or firm irregularity.
5. Evidence of hormone production.
6. Ascites.

One or more of the above findings may indicate laparotomy. If it is decided that laparotomy is not justified, further observation, often with the employment of hormone therapy, is the plan of medical management.

In the treatment of small polycystic ovaries or of the cystic ovary less than 6 cm. in diameter, cyclic estrogen or androgen therapy is indicated,* particularly in dysfunctional uterine bleeding.[14,23,24,35] Laparotomy should be avoided unless severe pain or uncontrollable hemorrhage occurs. If surgery is unavoidable, resection of the cystic areas with conservation of all possible ovarian substance is the accepted procedure. Only at the menopause—after the curettage—is roentgen therapy justified.

The life span of the corpus luteum is prolonged and cyst formation occurs in certain instances. This is presumably due to long continued production of luteinizing hormone by the anterior pituitary. A persistent corpus luteum results, and menstrual delay and prolonged and/or profuse dysfunctional bleeding, plus irregular shedding of the endometrium, are produced.

Curettage is indicated when uterine bleeding is excessive, prolonged or repetitiously abnormal. Average function often follows this treatment alone. However, we administer estrogens or androgens cyclicly to limit the effects of progesterone in recalcitrant cases.

Conservative surgery may be overdone, but is often feasible even with extensive pelvic inflammatory disease. Falk[12,13] has shown repeatedly that tubal division for salpingitis, with the retention of even a damaged ovary and dilated tube, will retain ovarian function for many years with very little discomfort.

MANY surgeons routinely remove the ovary on the side of a tubal pregnancy whether it is diseased or not. This is usually for convenience or ease in technic. The argument is that such ovaries are poor specimens if the tube is excised. Some of the arcuate vasculature to the ovary is necessarily divided. However, the retention of the ovary after laparotomy for ectopic pregnancy is generally entirely successful.[41,48] Many of these patients are also infertility problems and conservatism enhances the possibility of future conception.

Even with endometriosis, the powder-burned areas and contracted, scarred blood cysts can be excised by wedge resection and a respectable ovary may be retained.[5] Dermoids can be excised with closure of the capsule and retention of active ovarian tissue.

It is a good practice to aspirate ascitic fluid immediately upon opening the abdomen, prior to handling of a large or suspicious ovarian cyst. This fluid should be stained for malignant cells, and if cancer elements are diagnosed, x-ray therapy can be given without delay despite the grave prognosis.[10]

Ovarian cysts should never be aspirated at surgery for fear of spill. However, every ovarian cyst should be opened at the operating table to note the presence of papillary or solid zones. Benign, neoplastic, usually papilliferous cysts should be removed with or without the tube depending upon the desire for pregnancy. Since serous and pseudomucinous cystadenomata often become malignant, excision of the other ovary and tube in the woman over 45 years old with a truly suspicious cyst greater than 4 cm. in diameter is justified. Panhysterectomy is likewise required if cancer is definite or likely.

A number of operations have been devised for improving ovarian function or for reducing ovarian cysts. Puncture of ovarian cysts by a needle or scalpel is common practice. *This is a*

**Estrogen therapy:* Diethylstilbestrol, 1.0 mg. q.d. for 20 of 30 days cyclicly over 2 to 3 months.

Androgen therapy: Methyl testosterone, 10 mg. (sublingually) q.d. for 20 of 30 days cyclicly over 2 to 3 months.

totally worthless gesture. The cysts promptly reform if the situation is not further improved. Ovarian cystectomy has been popularized by Bonney.[4,6] The decortification operation of Raycraft[37] is still an experimental procedure. The splitting operation of Bailey[2] and Stein[46] and the Weed[50] wedge excision are likewise unproved in their results. Most of these authors avoid an explanation of why incision or wedge resection improves ovarian cyclism. It is agreed that certain of these operations may benefit ovarian function by increasing the circulation and by division of scars within the ovary. These latter procedures are rarely indicated. We can endorse Bonney's and Jacobson's careful excision of cystic structures when indicated. However, we believe that operations such as the Stein procedure should be considered as innovations. Since clinical indications for these procedures are vague, they cannot be recommended at the present time.

Ovarian suspension is not advocated. Almost invariably, considerable tension will be exerted or actual kinking of the infundibulopelvic ligament will leave the ovary much worse than if nothing were attempted.[11] If elevation must be effected, a vaginal pessary may help or suspension of the uterus may be required.[14,50] Whether to remove one or both ovaries in patients past the menopause is still debated. We note by vaginal smears that ovarian function may continue for years after the menopause. Middle age alone is not the best yardstick for ovarian excision. Some state that ovarian cancer may form in such inactive tissue.[9,45] It is true that we must be cancer-minded. However, if we were to remove tissue which might under certain unknown circumstances some day become malignant, we should be performing routine thyroidectomy, bilateral mastectomy, total gastrectomy and pelvic evisceration operations for prophylactic reasons in every woman over 40 years of age.[10]

SUMMARY AND CONCLUSIONS

1. Physiologic ovarian cysts are contrasted with those of a pathologic type and the differential diagnosis stressed. The malignant potentials and characteristics of neoplastic cysts are reviewed.

2. Certain functions of the normal and also of the cystic ovary are discussed and conservation of normal ovarian tissue for full function is advocated.

3. Causes of ovarian pain and dysfunction, particularly when associated with ovarian cyst formation, are reviewed.

4. Modern concepts of reasonable, adequate, conservative medical and surgical treatment of the various non-neoplastic ovarian cysts are presented.

5. Complete pelvic surgery is recommended in the treatment of certain precancerous or malignant ovarian cysts, but in general, unnecessarily radical ovarian surgery is decried.

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Diagnostic Clinic

Treatment of High Diastolic Type of Essential Hypertension

COMBINED USE OF THIOCYANATES AND SYMPATHECTOMY

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In 1934 the late Dr. Herbert Barker and I began to study patients with essential hypertension and we established criteria which we have now followed for 16 years. We believed that if we could choose a very definite group of patients with essential hypertension and define those cases so that everyone would understand what we were talking about, eventually we might make some contribution to an understanding of the mechanism of essential hypertension and also to its treatment.

We chose a group of patients whom the men at the Northwestern University clinic grouped as patients with a high diastolic type of essential hypertension. This meant that these people had a systolic blood pressure usually over 200 and a diastolic blood pressure over 100; that they showed a very progressive course which usually ended fatally; that they had no evidence of renal damage; that they had a high total protein and often a very high blood cholesterol, and that they were in contradistinction to the patients with hypertension whom the men at our clinic had classified as having the fluctuant type of hypertension. All of you recognize fluctuant hypertension as occurring in young individuals whose blood pressure may be very high at one time, low at another, but who for the most part go on about their business, lead perfectly normal lives and die of some other disease.

There is arteriosclerotic hypertension and the hypertension which occurs in women during the menopause. There are also patients with the high diastolic type, and finally that group of patients, which we have not found to be very large, who die very quickly and very promptly from a malignant type of hypertension.

We chose for surgical therapy, therefore, the high diastolic type because Dr. Barker and his colleagues in the medical clinic have been able to treat all of the other types of hypertension quite successfully by the use of the thiocyanates.

I think in this country Barker perhaps has done more work on the use of thiocyanates—or had up until the time of his death—than anyone else, and his work has been corroborated elsewhere, particularly in Great Britain and in other clinics where the thiocyanates have been administered carefully and where thiocyanate levels have been taken accurately and at frequent intervals. Barker found that many of these people, as a matter of fact the majority, could be treated with the thiocyanates if their blood levels were kept accurately. Further, there

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were individual variations of the height of the blood level necessary to maintain a proper pressure and a proper clinical state of well-being; the thiocyanates reduced the blood cholesterol and the total blood proteins. So the majority of about 1000 patients have been treated medically by proper administration of thiocyanates.

There was a group with the high diastolic type of hypertension who did not do well under thiocyanates because it was necessary to raise the thiocyanate level so high that the patient did not tolerate the drug; nausea and vomiting were produced, sometimes diarrhea, and the blood level could never be raised high enough to get a good clinical result. Most people think that because the thiocyanates do produce those symptoms, they are toxic drugs, but we have proved in our laboratory that one can raise the blood thiocyanate level in animals—and we have also proved it in man—by giving the drug intravenously and not producing nausea and vomiting and diarrhea. Although there is a gastrointestinal intolerance in some people for this drug, the blood level can be raised so that it is high enough to get an effect, if there will be one, without producing gastrointestinal symptoms.

Therefore, we chose for surgical treatment this group with high diastolic hypertension which was resistant to the thiocyanates. We did this by accident. Dr. Barker had a patient who had been treated with thiocyanates rather unsuccessfully. The man became disturbed because he wasn't getting better, went to the Mayo Clinic where he was refused operation the first time and then came back to Chicago. Here he was again treated with thiocyanates, rather unsuccessfully because his blood level could not be pushed high enough, and he again went to the Mayo Clinic. This time a thoracolumbar sympathectomy was done with a temporary decrease in his blood pressure, but by the time he got home his blood pressure was right where it had been when he left. Rather in desperation Barker gave him the thiocyanates again, and much to his amazement he not only controlled his blood pressure but all of his clinical symptoms, his blood cholesterol and his total protein, on a rather low blood level of thiocyanate.

Barker's experience started us on the study of this whole problem. In other words, why did this individual become sensitive to the thiocyanates, or rather why did the thiocyanates become more effective after sympathectomy?

We have operated upon a very small group of cases, so if you would expect to come to some conclusion from what I have to say simply on the number of cases you will be disappointed, and I am rather proud of the fact that we have operated upon so few. We have now operated upon 100 patients with a high diastolic type of hypertension who have not done well with thiocyanates. In none of those patients have we been able to affect the blood pressure permanently by any type of sympathectomy. We have done thoracolumbar sympathectomies of varying extents, and regardless of the extent of the operation, the operation alone has not affected those patients' blood pressures. They have had a postural result—when they stood up their blood pressure was lower—but permanently that effect has never lasted, and as I said, these patients had not responded well to the thiocyanates.

The majority of those patients have done very well following sympathectomy when the thiocyanates have again been given, and they have been carried on a lower blood level of thiocyanates. Clinically I believe, and I am sure my medical colleagues at Northwestern and

Passavant agree, that there is a group of high diastolic essential hypertensive patients who do not do well with properly controlled thiocyanate therapy—very careful blood levels varying with the individual—but who have done extremely well when operated on and given thiocyanates.

Naturally you will want to know, as we did, why this occurs. Along with our clinical investigation upon these patients, we carried on an experimental investigation to try to find out why the thiocyanates act so well after a sympathectomy; as a matter of fact, why they act in patients wih hypertension who have not had a sympathectomy. We have been able to produce a hypertension in dogs with the Goldblatt clamp on the renal vessels—the closest approach we can get in the experimental animal to the hypertension which occurs in human beings—without evidence of kidney damage, and we have been able to drop those high blood pressure levels in the hypertensive dog by the administration of the thiocyanate. We have been able to prove that we need less thiocyanates in these animals if we perform a sympathectomy, so that we have been able to carry on experiments upon animals to correlate the observations made in man.

Recently an hypothesis has been offered on the question of hypertension, particularly by Shorr and his coworkers in New York, which would balance the liver against the adrenal glands in hypertensive patients. In other words, Shorr and others believe that the liver produces a vasodepressor substance and that the adrenal glands produce vasopressor substances, and in hypertensive patients these are out of balance; the patient either does not have enough depressor substance manufactured by the liver or he has too much pressor substance which comes from the adrenal cortex. So we have taken this hypothesis into the experimental laboratory and to our patients.

If we examine the adrenal glands of a dog or rat to which we have given thiocyanates, depending upon the level of those thiocyanates in the blood we can demonstrate a depletion of the lipid granules which occur in the three layers of the adrenal cortex. It is those lipid granules which have been shown by accurate cytochemical method to be related to the 17-ketosteroids. This cytochemical method is so accurate that when those lipid granules are depleted in the adrenal cortex one can conclude that the function of the adrenal cortex is thereby influenced. By giving thiocyanates to normal rats and to dogs, we can show depletion of these granules in the adrenal cortex, and the granules will reappear when the thiocyanates are discontinued.

We have repeated this experiment in hypertensive dogs, and we can show a pattern of depletion in the lipid granules of the adrenal cortex in the hypertensive dog; we can replace those granules when the thiocyanates are taken away from the animal, and we can produce a more marked depletion of those granules of the adrenal cortex when the animal has had a bilateral thoracolumbar sympathectomy.

We are able to show exactly the same microscopic appearance of the adrenal cortex in patients in whom we have taken a biopsy of the adrenal cortex when their blood has been free from thiocyanates and when they have had a very high blood level of thiocyanates. In other words, when doing a thoracolumbar sympathectomy on one side we take a section of the adrenal cortex when the patient's blood level is zero, and at the next stage, when we remove the opposite side and after we have raised that patient's blood thiocyanate level to 20 or 30 mg. per cent. We study his adrenal cortex and find the microscopic appearance of depletion of the lipid granules is exactly that which we see in the experimental animal.

We have done other experiments upon animals to try to find some explanation for hypertension, proceeding on the theory that the liver and adrenal cortex are out of balance in a hypertensive patient. What the vasodepressor substance is I don't know and I don't think anyone else knows. However, we have produced a mild chronic insufficiency of the liver in experimental animals by putting a clamp on the portal vein and partially occluding the vessel. These animals will live and we have had them alive for a year. Their blood chemistries reflect a mild liver insufficiency. The microscopic appearance of their livers is that of a mild fatty infiltration. The nuclei of the liver cells are perfectly intact, and when the portal vein occlusion is removed the fatty infiltration disappears and the liver looks perfectly normal again. The blood pressure in such an experimental hypertensive animal can be reduced permanently by such a procedure.

It is interesting to note that a patient receiv-

ing thiocyanates for a long period of time will show the same changes in his liver cells—a fatty infiltration without disturbance of the liver nuclei.

We can also show a change in the lipid distribution in the adrenal cortex when we have a partial occlusion of the portal vein, a depletion in all three layers, but the depletion pattern is different from that in the animal to which we have given thiocyanates. We reduce the pressure in the experimental animal by a partial occlusion of the portal vein, his pressure comes down and stays down, and we produce a change and depletion of the lipid granules in the adrenal cortex if we give him thiocyanates and take out his thoracolumbar sympathetic chain on both sides.

I believe it is wrong to operate indiscriminately upon everybody who has hypertension, because I believe that for the most part patients with hypertension can be treated successfully medically. There is, however, the group which cannot be so treated and it is toward this group we are pointing and trying to find out why the things we have done have worked.

Let me summarize our approach to the whole problem of hypertension in this way: We believe that the majority of patients with hypertension are patients who can be treated successfully medically. I think that would also be your experience. Certainly a patient who has a fluctuant type of hypertension, who is a young individual, might do very well when you remove part of his sympathetic nervous system. The patient with a menopausal type of hypertension, who gets better when the menopause is over, in our belief is not a patient who should be operated upon. We do not believe that operation does anything for a patient who has an arteriosclerotic type of hypertension, either. We think that individual can be treated successfully medically.

Our experience at the clinic at Northwestern with hypertensives has been that the thiocyanates have been the best and most effective drug when properly employed to treat a large group of hypertensive patients. Thiocyanate has to be employed correctly, careful blood levels must be kept all the time for that particular patient; his blood level may vary from that of another patient with equally severe hypertension. The high diastolic type of hypertensives for the most part will do well with the thiocyanates when they are properly controlled.

In the group of high diastolic hypertensive patients who do not do well with thiocyanates, and upon whom we have operated (100 of them), we have observed that the majority do well after a sympathectomy when they are given the thiocyanates again. The combination of thiocyanates and the sympathectomy has proved to be an effective treatment in that group of patients. Now why? Because from our experimental work and our combined clinical investigation we believe that the thiocyanates depress the function of the adrenal cortex, and we have shown the evidence for that in the depletion of the lipid granules in the adrenal cortex. We know that a sympathectomy depresses the function of the adrenal gland, and when you combine a sympathectomy with the thiocyanates you have a double depressor effect upon the function of the adrenal cortex, and so we believe they work in conjunction.

The other question regarding the participation of the liver in hypertension is an experimental one. We believe we have offered some proof that a partial occlusion of the portal vein reduces the pressure of an experimental hypertensive animal. It not only does that, but it also produces a depletion of the lipid granules in the adrenal cortex as do the thiocyanates, so that if it works in any other way, which is possible, it also produces a depression of the adrenal cortex.

One last word—and I think that our work for 16 years supports this statement, that the hypertensive patient should be approached surgically as a problem in which we may make some contribution to an understanding of the disease process, and that not all hypertensive patients who have a blood pressure of 160 or over should be operated upon.

Diagnostic Clinic

Diagnosis and Treatment of Polycythemia Vera

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Interest in treatment of polycythemia vera has outmoded careful diagnosis and study of the patient afflicted with this disease. Radioactive isotopes presently claim attention as the treatment of choice. The purpose of this clinic is to emphasize the importance of study and observation of the individual patient before deciding on the type of treatment to be employed.

I BECAME interested in polycythemia many years ago. When I went into practice, my first patient had polycythemia. At that time I was very much interested in treatment of the disease, and in association with Dr. S. H. Hurwitz[2] of San Francisco, I treated this patient with benzol by mouth and roentgen ray therapy over the spleen and long bones. She made very remarkable progress under this treatment; a remission followed lasting for 11 years. We did intend to publish her case as a cure, as we were very enthusiastic about the results obtained at that time. Owing to my habit of procrastination, we put off this report, and it is lucky that we did so because she relapsed between the eleventh and twelfth year of the disease. I had the rare opportunity of following this patient for 32 years, and she was a great inspiration to me in the study of this disease. About two years ago she died of monocytic leukemia.

After a number of years of study and experience with polycythemia vera, I have come to the conclusion that we emphasize too much the treatment and not enough the nosology and natural history of this disease, and particularly have we lost sight of the idea of investigating its cause. There is no more, or little more, known about the cause of polycythemia vera today than there was at the time of the original description by Vaquez and the subsequent description a few years later by Osler.

We have 2 patients here who illustrate types of this disease. I wish to emphasize that we should study patients carefully with relation to the type of disease, whether it be mild, moderately mild, or whether it be a more severe type of polycythemia vera with thromboembolic phenomena.

The first patient, who was kindly supplied by Dr. LeRoy Sloan, is very interesting because apparently in the beginning there was more attention paid to some of the other features of his disease, namely, the condition of the arteries, and the pain he had in his abdomen which was thought to be due to gallbladder disease. He also was found to have a duodenal ulcer, and considerable treatment was given him for that before it was discovered he had polycythemia.

One does not notice particularly now that he has the ruddy cyanotic complexion that is so typical of these cases, but that is not always present. I think it is a mistake to feel that you can diagnose these patients by simply looking at them. Some of them you can, it is true, but some you cannot. However, there is one very important point which I should like to call to your attention: that is, what is known as acrocyanosis—the cyanosis of the lobes of the ears, the nose, and very often of the eyelids; if you

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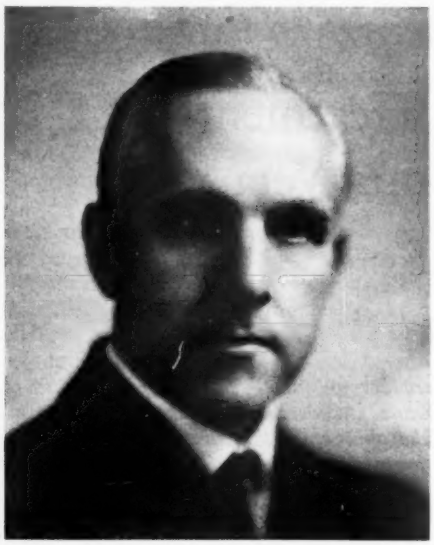

ERNEST H. FALCONER

turn the eyelids down you will notice they are very deeply congested. We have that condition here. I have just examined this patient, and he has a very dark, congested appearance of the conjunctiva, also of the gums. (To the patient): Do you have your own teeth?

PATIENT: No, I haven't.

DR. FALCONER: That very often happens with these patients and there is a little story that goes with it which I will mention later, namely, about the difficulties and dangers of teeth extraction in such patients. Very often, though, in patients with polycythemia vera the gums have a peculiar appearance which is quite diagnostic. It is not simply a cyanotic gum—it has a peculiar reddish cyanotic hue that extends well down to the base of the gum.

At present this patient presents a picture more of arteriosclerosis with probably a coronary block and angina pectoris. Angina pectoris is not common in these patients, according to my observations, but coronary block is fairly common, with the subsequent myocardial infarction.

At one time you were a switchman?

PATIENT: That is right.

DR. FALCONER: That is pretty heavy work, I should say, a little hard on the cardiovascular system. At the present time he has not been able to do work of any kind because exertion brings on pain. You take a fair number of nitroglycerin tablets?

PATIENT: I do.

DR. FALCONER: Another interesting feature in this patient is his peripheral vascular disease, which is obviously present. He has a great deal of trouble with paresthesia. I will discuss this subject later.

I have examined his limbs and he does have, I think, very definite evidence of peripheral vascular disease.

He is on radioactive phosphorus at the present time. His hematocrit is still a little high; it is about 49 now, which is higher than we like to keep it in the peripheral vascular complications of this disease.

THE next patient is interesting largely from the standpoint of thromboembolic phenomena. He is having a great deal of difficulty with his circulation, particularly in the extremities. He has apparently had embolic phenomena in the vessels of the arms as well as the feet. He has had ligation for varicose veins; he has very extensive varicosities. I present him at this time largely because of these vascular complications, which I think are tremendously important in connection with this disease. He had, in the beginning, a rather high red cell count, running up to over 8,000,000. His hematocrit was 67, which is quite a bit above normal. He has been on DICUMAROL® at different times, but it had to be discontinued, apparently because of hemorrhages, particularly from the kidneys.

The diagram in Figure 1 is borrowed from an article by Dr. Russell Haden.[1] It shows so well the laboratory findings in polycythemia vera that I copied it from his illustrations. The main thing to emphasize, I think, is the large mass of red cells in the hematocrit, which is 65 per cent in this particular case. The red cell mass is what Haden calls the erythron. The erythron is greatly increased in polycythemia. Normally, the red cell pack is about 45 per cent and the rest is plasma, 55 per cent. The important thing is the relation of the red cell mass to the body weight, which in males is about 30 cc., in females 27 cc., per kilogram. This is particularly important because in symptomatic polycythemia the number of red cells per cubic centimeter may be considerably increased, but

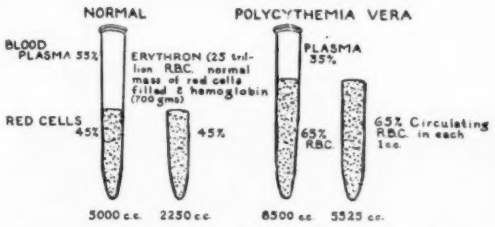

FIGURE 1. Red cell mass in normal persons and in those with polycythemia vera.

you do not find that the erythron, the red cell mass, is greatly increased in proportion to the body weight. Of course, the blood volume in the symptomatic type of polycythemia is not increased, whereas in polycythemia vera it is greatly increased. This increase is due mostly to increase of packed red cells and not to an increase of plasma.

So much for the laboratory findings of this disease. I think in all instances, even though you strongly suspect polycythemia from the clinical examination of the patient, the diagnosis has to be confirmed by laboratory findings and, in the last analysis, by the blood volume. Although a very high hematocrit may suggest the diagnosis and, along with the clinical findings, might be sufficient to enable one to make a diagnosis in these cases. It is particularly important, when you put the patient on treatment and you are following the blood count, to follow the hematocrit rather than the red cell count in the counting chamber, because in the counting chamber mistakes are very often made in the enumeration of these cells.

Now I want to discuss our first patient, because he brings up a great many of the features which we see in various types of polycythemia vera. For example, duodenal ulcer is a fairly common complication, certainly much more common in patients with polycythemia than in the population as a whole. Just what the cause of it is, is difficult to say; I believe the more recent feeling is that the ulcer is due to endarteritic changes in the vessels about the duodenum and that the gastric juice digests the mucosa which is impaired by insufficient circulation. However, I don't think the actual cause of duodenal ulcer has ever been accurately demonstrated.

Angina pectoris, as I stated before, is not commonly associated with polycythemia in my experience. Perhaps it is in the experience of others. Coronary block, coronary thrombosis, along with other types of thrombosis of arteries, is fairly common. As a matter of fact there may be a block in any of the arteries in the body in these cases, and one should always be on the lookout for such phenomena when treating these patients.

I do not know whether the incidence of anginal attacks can be decreased by depleting the blood volume, by getting it down to normal, but I should imagine that one could do so. However, if you have a patient with a coronary block it is very important, even if the red count and hemoglobin are not much above normal, to treat that patient promptly by venesection and diminish the blood volume.

The pain which the first patient had—he was later operated on for gallbladder disease—is interesting, because we noted in the patient that I mentioned having followed for 32 years[2] great pain of sudden onset in the right upper quadrant of the abdomen. This followed an 11-year remission. Although her physicians could not demonstrate any evidence of gallstones or dysfunction of the gallbladder by roentgenographic studies, still they felt that the gallbladder must be causing her symptoms and they were very anxious to remove it. When they were told about my experience with this patient after having followed her for a number of years, I was able to persuade them not to operate but rather to do a venesection, and later to put her on phenylhydrazine and the whole affair cleared up, with no return, fortunately. I believe these attacks must be due, in these cases, either to a thrombosis of a branch of the hepatic artery or possibly to a block in a branch of the portal vein itself.

The first patient I presented has a very interesting situation with regard to his paresthesia which I think is of considerable importance in these cases. While it is difficult to explain and it is very often difficult to find very much in the way of objective symptoms to bear out their complaints of paresthesia, yet it is very real and gives the patients a good deal of trouble. I believe it always has to be taken into consideration in the treatment.

The second patient has had the embolic phenomena which are so characteristic of the disease, and he has peripheral vascular disease of the extremities, which I think is both interesting and important. For some years, we have

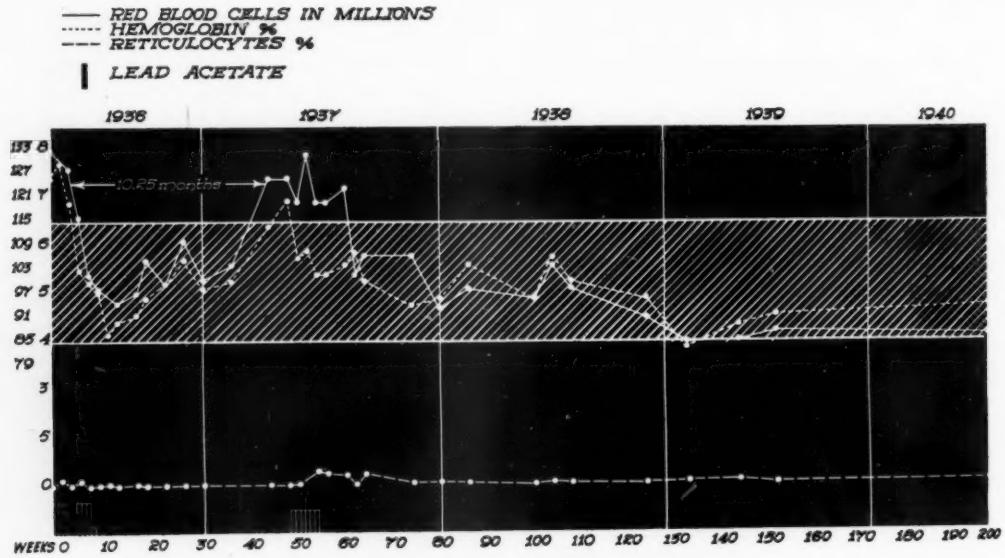

FIGURE 2. A remission of more than six years following lead compound by mouth.

been conducting studies with the peripheral vascular clinic at the University of California and recall 3 patients very distinctly who had peripheral vascular phenomena with a slight increase of their red cell count, but investigation did not disclose a distinct polycythemia. Yet as we followed these patients, in one instance for three years, very definite polycythemia vera developed, as shown by an increase of blood volume and of red cell mass, and by the signs and symptoms of polycythemia vera.

As a rule, these patients do not have very markedly enlarged spleens. The second patient I showed you does have an enlarged spleen. The first one does not—at least there is no mention of a palpable spleen. The first man might fall presumably into the category of cases described by Geisböck—those occurring in older individuals with arteriosclerosis where there is usually hypertension, maybe some actual cardiovascular involvement, and no enlargement of the spleen. He calls these cases polycythemia hypertonica. They are known also by the name of Geisböck's disease. I do not, however, subscribe to this classification because one not infrequently finds, in following these patients from year to year, that they do later show an enlarged spleen. The second case does have an enlarged spleen, but it is not tremendously enlarged, and it has been my experience that patients with thromboembolic phenomena are not so apt to have very large spleens.

In this connection, however, I would like to say a word about enlarged spleens, especially the tremendously enlarged spleens that you occasionally encounter. You may be tempted to have them removed in order to get better results from treatment because, as a rule, in my experience, patients with very large spleens do not respond well to any type of treatment, even radioactive isotopes.

In one instance we tried to remove the spleen from a patient whose spleen was very large, and in whom we discovered by splenic puncture before operation that his spleen was almost entirely filled with platelets and megakaryocytes. We were very much disturbed by this finding because we felt that he would probably have embolic phenomena if operated on, so we gave him dicumarol. But after operation he developed what we thought were emboli and we discontinued the dicumarol on account of hemorrhage. The patient did not survive and subsequent postmortem examination showed that he did not have emboli but that he really bled to death into the peritoneal cavity. So we had this curious situation of a man with a very high platelet count, the spleen filled with plate-

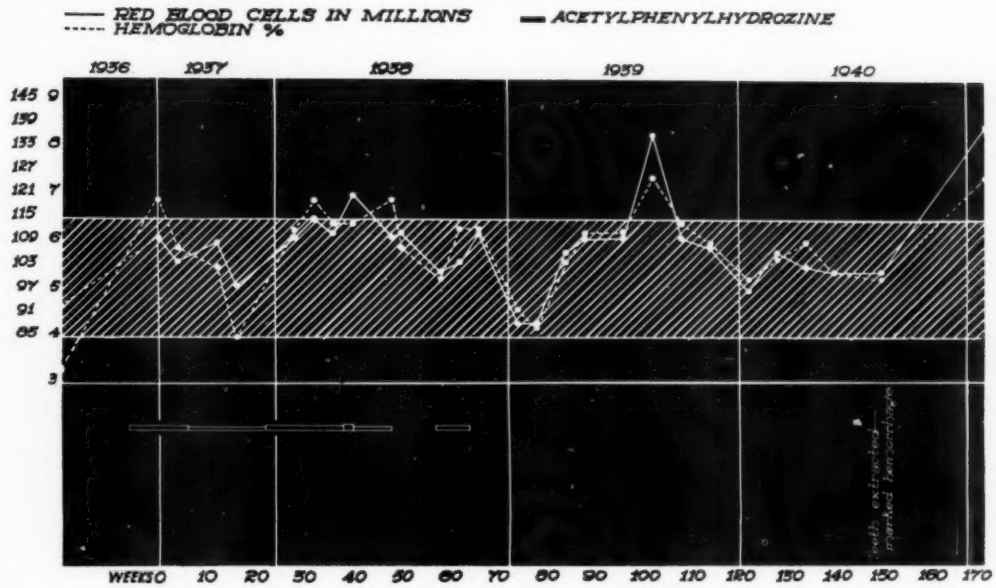

FIGURE 3. Satisfactory response to small doses of acetyl phenylhydrazine over a period of 19 years.

lets, and yet he literally bled to death as a terminal event. I would not advise removing the spleen in these patients. It usually has resulted disastrously.

I would like to emphasize that no one type of treatment is always successful and that there are many different types of treatment that can be used for these patients. For example, Figure 2 represents treatment with a lead compound. I was interested some years ago in using colloidal lead phosphate in these patients because we had been studying the effects of lead on the bone marrow and blood, and accidentally discovered that it was quite efficient in reducing the blood of polycythemic patients.[3]

This man had a very high hemoglobin and red cell count when he came to us. His hemoglobin was up to 135. I put him on lead acetate by mouth following which he had a prompt drop in both red cells and hemoglobin. A remission followed which lasted a little over 10 months, when his blood began to rise again almost to the original height. I put him on lead acetate again—not a great amount—3 gm. by mouth, in 1937. Following this he had his second drop and remained down in 1938, 1939 and 1940, when we lost track of him and did not see him again until about 1944. He had returned to the San Francisco Bay region and was working in the shipyards. We did some blood counts on him at that time and found that he was still normal in 1944, a remission of approximately six years.

FIGURE 3 is a very interesting graph of a woman treated with phenylhydrazine.[3] I think this shows that in certain cases phenylhydrazine is a very good form of treatment. As a matter of fact, this patient was treated with acetyl phenylhydrazine. She was sent to me by Dr. Charles Stone of Galveston, Texas, who with his associates had been using acetyl phenylhydrazine instead of phenylhydrazine because they thought it was less toxic.

This woman came to me first in 1936. I followed her over a period of about 12 or 13 years, and Dr. Stone followed her for at least six years before she came to me. You will note how easy it is to maintain her blood at a normal level with very small doses of phenylhydrazine, 0.1 gm. once a week. Where you see a slightly thinner line, the dose was practically 0.1 gm. about once every two weeks or possibly, in some instances, once every three weeks. You will note that she got along very well. In 1940 we had a couple of her teeth pulled that were very loose in the upper jaw—they felt as if you could pick

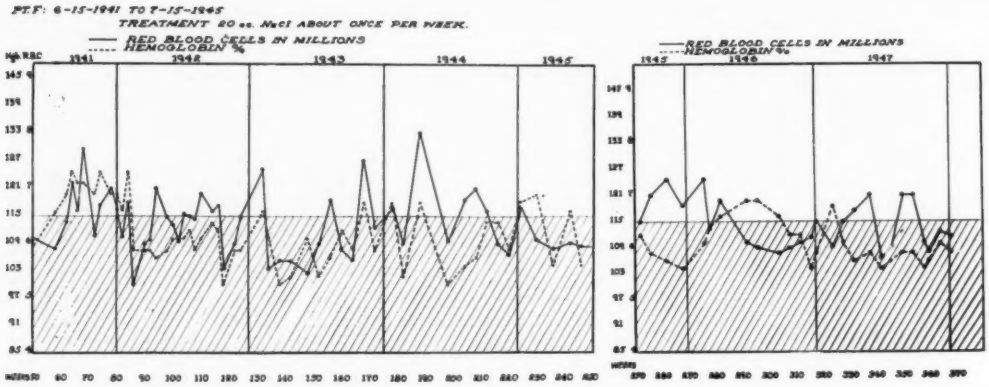

FIGURE 4. This patient has been under observation for 10 years. The first year he received 10 cc. normal saline once per week; thereafter for 9 years, once per month.

them out with your fingers. However, I insisted that she go to the hospital. She very unwillingly went. We called in a very good dental surgeon to do the extraction. It was very easy to remove these two teeth, to get them out of their sockets, but within 24 hours she started to bleed. She had a tremendous hemorrhage which infiltrated down into the floor of the mouth. The hemorrhagic area became infected, and then she developed Ludwig's angina, the infection spreading down into the mediastinum with a bilateral bronchopneumonia supervening. She was in the hospital about two months altogether, but she recovered and we continued our treatment with acetyl phenylhydrazine. She only recently passed away, at 75 years of age, after 19 years of this treatment. So for this particular patient, acetyl phenylhydrazine was as good a form of treatment as one could have employed.

A very interesting man in the series we have been following has what comes the nearest to emotional polycythemia of any case that I have seen (Figure 4). When he came to us first he had been driven out of Germany into Holland and then from Holland to the United States. He is a Jewish refugee, and was in a tremendously upset emotional state as he had been very wealthy in Europe and had lost most of his wealth. When we saw him first in 1941 he had an undoubted polycythemia; his blood volume was up about 7000 cc., and the packed cell volume was very high, over 4000 cc. He was sent to me by one of my colleagues who had tried him out on several different types of treatment. When he came to me I decided to watch him and study his disease for a time, giving him only normal saline. I gave him 10 cc. of normal saline by vein and, strange to say, he did not complain at all about any adverse symptoms. I tried to withdraw the normal saline but he would have none of it, so he has continued to receive nothing but normal saline for 10 years, and during the last five years his blood very seldom has risen above normal. Sometimes it goes up a little, but it comes down again, and he has been on no specific therapy to reduce the blood level except normal saline. Suppose he had been getting radioactive phosphorus. That certainly would get the credit for his improvement, but he has not had any radioactive phosphorus whatever.

In summary, emphasis should be placed on study of each patient with polycythemia vera for at least two or three months before deciding on the best type of therapy for the individual needs and comfort of that particular patient. If indications arise for immediate treatment, venesection can readily be carried out. As no therapeutic agent up to date can be said to cure the patient, it would appear rational to question whether any single therapeutic regimen is the best for all cases of polycythemia vera, regardless of type or severity of the disease, in the individual patient.

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Diagnostic Clinic

The Significance of Frequency of Urination

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THE patient I wish to discuss, a female age 65, was admitted to the Presbyterian Hospital on October 29, 1950. Her present illness began four weeks before with severe frequency and urgency of urination. She was obliged to void every hour or every hour and a half. The frequency was partially relieved by hot vaginal douches and sitz baths. During the attacks she has pain in the "middle of her back."

The family history revealed that one brother died of diabetes at 47 and another brother died of prostatism and hypertension.

The examination of her urine in the office of her physician and in the hospital showed the presence of pus (voided specimen) and a diagnosis of cystitis was made. When I elicited her history, she stated that she consulted a urologist more than 20 years ago because of frequency of urination. The urologist found sugar in her urine and referred her to an internist for further treatment.

The physical examination upon admission to the Presbyterian Hospital was negative except for the presence of a suprapubic scar, the result of an abdominal hysterectomy for fibroids. The blood count, Kahn test, blood chemistry, x-rays, and intravenous pyelograms were all negative. Two cultures of her urine were sterile. Examinations of the stool for amebae were negative. The electrocardiogram showed a right bundle branch block and chronic coronary disease.

On several occasions before this assembly I have stressed the importance and value of obtaining a careful detailed history. The physician himself is the one to evaluate the history. The importance of this factor is illustrated by the present case.

This patient stated that she saw a urologist 20 years ago because of frequency of urination and that sugar was found in her urine. She has been on diabetic management ever since. Let us bear in mind the fact that her symptom, i.e., frequency, has been present for over 20 years.

A diagnosis of cystitis was made, based on the symptoms and the presence of pus in the urine. Cystitis in women is a rare disease. However, the symptom complex which leads to the diagnosis is relatively common.

The flat films and the intravenous urograms were absolutely negative.

My first question was whether or not the pus was found in a voided or catheterized specimen. I was told the pus was found in a "clean specimen." I assured them that I did not take much stock in a so-called clean specimen, and that in women only a catheterized specimen should be examined. If a voided specimen is negative, all well and good, but if it shows pus one should examine a catheterized specimen.

After a diagnosis of cystitis is made, the patient as a rule is given one of the sulfonamide compounds or one of the antibiotics, or both. It is to be remembered that the treatment should not be carried out too long. If it fails to relieve the patient's symptoms within a reasonable time, a complete urologic study should be made.

Let us now consider the fact that this patient has had attacks of frequency of urination for more than 20 years. We might rightly ask:

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HERMAN L. KRETSCHMER

What type of lesion could she have that would produce these symptoms for over 20 years and yet have her looking hale and hearty?

If the symptoms were due to carcinoma of the bladder, she would not be alive today. If the frequency of urination were due to urinary tuberculosis, there would be no periods of remission such as she has had, as she is free of symptoms between the acute attacks. Likewise, stone in the bladder does not have periods of remission.

From the long duration of the symptoms, one at once thinks of the possibility of elusive or Hunner's ulcer. In these cases there are no periods of remission and the patient is obliged to void many times at night.

In my patient there is no history of nocturia. The fact that she has great frequency during the daytime and rarely arises at night to "pass her water" is very important and significant.

Let us now consider some other possible causes for her symptoms. In many women with so-called cystitis, the primary focus is located in the intestinal tract. A careful history reveals the presence of a severe constipation, the use and abuse of cathartics, attacks of diarrhea, or a history of a spastic colon. These factors were absent in the patient now being discussed.

Frequency may be due to lesions in distant parts of the body such as the frequency associated with hyperthyroidism. I recently saw a patient with great frequency of urination who was taking three grains of thyroid a day. This easily explained her frequency.

Organic disease of the central nervous system may be responsible for frequency of urination. I refer to such lesions as tabes dorsalis, pernicious anemia, multiple sclerosis, etc. The diagnosis in this group of cases offers no special problems. Some of these patients suffer from cardiorenal-vascular disease, the recognition of which should be relatively easy.

Lesions of the gynecologic tract are often responsible for frequency of urination. None were found on a very careful pelvic examination.

One of the common and yet frequently overlooked lesions in women is chronic urethritis. This lesion is one easily recognized. It was absent here.

The x-ray films and the intravenous urograms were negative, as I have said. The cystoscopic examination and ureteral catheterization as well as the urinary examination, urine cultures and examination for tubercle bacilli were all found to be negative.

The diagnostic problem here concerned itself with the cause of her symptoms, there being no organic disease present to account for them.

As I have stated, the examination of her nervous system revealed no organic disease. Therefore, psychiatric consultation was requested. I obtained her consent for psychiatric consultation after I explained to the patient that I was unable to demonstrate the presence of any organic disease to account for her symptoms, that I thought her trouble was due to her nerves and that I would like to have a "nerve man" see her. To this she readily consented.

This examination revealed the fact that she was very unhappily married. Her husband's sister came to live with them many years ago. The patient stated that her husband was much more interested in his sister than he was in the patient. This sister is an "old maid." So in this case we were dealing with a psychic problem and one that needs psychiatric and not urologic care. Hence she needs no sulfonamide compounds, antibiotics, vitamins, hormones, or local treatment. Once the diagnosis was established the treatment was obvious, as were the things not to do!

Diagnostic Clinic

Oral Health in Children

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Many articles on oral health in children have appeared in dental journals with relatively few in the more commonly read pediatric literature.

By various surveys it has been shown that 92 per cent of the population requires dental care. It has also been shown that 50 to 60 per cent of children under five years of age require active dental care. The problem is so great that the dental profession alone cannot be expected to take care of this great load. Because of this the medical profession must become interested in the problem. To do so, they must know the accepted theories of caries initiation as well as the principles of oral health and have a realization of what good dental repair should comprise.

This paper reviews the present accepted methods of the control and prevention of dental caries as well as evaluates the value of dentifrices.

The past decade has seen a continuously increasing emphasis on infant and child care. General practitioners and pediatricians are in the forefront of this campaign. Infant mortality has been drastically lowered; contagious diseases are on the decrease because of the commonly accepted immunization procedures; many congenital anomalies are now corrected by advances in surgery; behavior and emotional problems, though still prevalent, are better understood and handled; public health and hygiene have become an accepted part of most school and community programs—to name only a few of the effects of this campaign.

It is now becoming evident that we, as physicians, must also become interested and assist in the field of oral health. It would seem that most of us either have not been aware of the enormity of the problem or have been too content to let the dentist shoulder the entire responsibility.

Many articles on oral health in children have appeared in dental and public health journals but relatively few have appeared in the most commonly read general medical and pediatric literature.

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THE PROBLEM

In this discussion on oral health I would like to take up only that part which pertains to the teeth. If one considers dental caries a disease of the calcified tissue of the teeth then one must realize that dental disease is the most prevalent of all disease processes affecting children. It is no respector of age, income or special groups.

It is estimated that 92 per cent of the population requires some dental care. McCall,[1] in 1938, found that about 50 per cent of 2-year-old children examined at the Guggenheimer Clinic in Boston had one or more carious primary or deciduous teeth and that 60 per cent of 5-year-olds had 7 or more cavities and one or more teeth needing extraction. A study at the University of Iowa[2] showed that the caries incidence was 1.1 at 2 years of age, 2.7 at 3 years, 4 at 4 years and 4.1 at 5 years of age. There are about 10,500,000 children under 5 years of age in this country and about 2,000,000 children reach the 2-year-old level each year. From this one can estimate the tremendous problem when one con-

JOHN A. BIGLER

siders that there are only about 70,000 dentists.

In our own pediatric clinic at the Children's Memorial Hospital we have about 8550 children registered. It can be estimated that about 7500 of these are potential dental patients 2 years of age or over. It can also be estimated that a bare minimum average of three hours a year is needed for each child to keep that child in good dental health. This would involve about 23,000 dental hours a year which would keep a staff of 11 to 12 dentists working 40 hours a week for 52 weeks. Such figures are staggering and make one realize that the problem is not one for the dental profession alone.

All of us have an obligation to society and this obligation must be borne equally by the physician and dentist. Pediatric dentistry is making very rapid advances. Medical care should include examination of all the structures in the mouth instead of just the tongue and the tonsils. There has been poor observation when no mention is made of the condition of the teeth, gums, gum boils, malocclusion, etc. The physician should be so well informed on oral health that he can give intelligent advice on hygiene, prophylaxis and adequate dental care. The physician is in an advantageous position because of his frequent contact with both patient and parent. He can initiate and train both parent and child in accepted methods of oral health. The problem of dental caries is so great that prevention and control are as important as dental repair.

WHAT CAUSES DENTAL CARIES?

To participate in a program of oral health one must know the accepted theories of caries initiation. Dental caries is an environmental process and the theories of its initiation are as follows:

1. Inclusion of excess fermentable carbohydrates in the diet.
   a. They are often soft and sticky foods which are retained about the teeth that are not self-cleansing. This furnishes a substrate for the growth of bacteria capable of initiating processes that cause tooth destruction.
   b. They may serve as substrate for forming acids that cause decalcification.
   c. They may offer an optimal bacterial enzymic action.
   d. They may cause an increase in the growth and metabolism of bacteria that act deleteriously on the teeth.
   e. They may dilute the diet so that the protective food intake is decreased.
2. The presence of an increased number of bacteria in the oral cavity, such as the *Lactobacillus acidophilus*. They act on the tooth enamel to hydrolyze glycoproteins, releasing an acid which causes tooth decay.
3. Changes in the physical, morphologic and chemical characteristics of enamel surfaces.
   a. Retention of food between the teeth because of malocclusion due to its many causes.
   b. Maternal diet.
   c. Deficiency diseases, infections, etc., during the formation of the teeth.
   d. Heredity.
4. The protection of saliva due to increased amounts and some protective substance it contains.

The importance of fermentable sugars to dental caries is a strongly advocated theory at the present time.[3,4] It is felt by the proponents of this theory that the fermentable carbohydrate acts as a substrate for acidogenic bacteria and their enzymic systems which then form acids which act upon the tooth enamel causing decalcification and initiation of caries. It has been demonstrated that when excess fermentable sugar is consumed, the lactobacillus count in the mouth is high.[5,6] It is stated that the lactobacillus count will serve as an index to the amount of dental caries that can be expected.

Pincus[7] and Atkinson[8] have reported that the

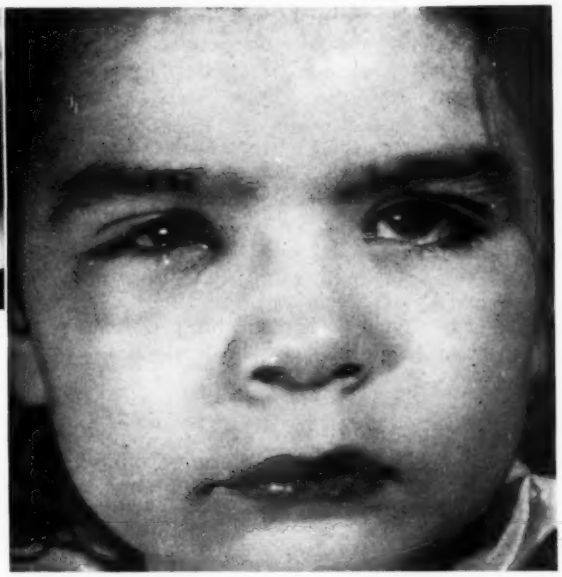

FIGURE 1. Broken deciduous tooth with apical abscess in five-year-old girl.

FIGURE 2. Swelling of right upper lip and right side of face from apical abscess and cellulitis from a deciduous tooth. Same patient as in Figure 1.

bacteria which are increased in high carbohydrate diets act directly on the glycoproteins of the tooth, hydrolyzing them to form acids which then cause decalcification of the tooth.

Within one and one-half minutes after taking a fermentable carbohydrate in the mouth the reaction on the teeth becomes very acid—the *p*H falling from 7.0 to 5.0. The taking of fermentable carbohydrates at mealtime is not as harmful as between meals because of the dilution and washing that occurs by other foods and fluids. It is not only a matter of how much sugar, but also of the frequency and the time it is eaten.

THE average American has a very "sweet tooth." It has been estimated that 40 pounds of sugar a year should supply the necessary carbohydrate calories, but the average consumption is now about 142 pounds per person, which is higher than in any other nation. Consider the large amount of candy, gum, soft drinks and other high sugar confections consumed by children between meals. With so much sugar consumption it would seem probable that dilution of the diet would result with a smaller intake of protective foods.

Physical changes in the mouth are also of importance. Malocclusion may cause an abnormal retention of food about the teeth and poor mastication. It may result from mild or severe congenital abnormalities in the development of the teeth or jaws, absence of teeth, marked dental caries, thumb sucking and mouth breathing.

Sognnaes,[9] in working with small animals, reported that if the maternal diet was natural during the development of the offspring's teeth then dental caries did not develop in the offspring when fermentable sugars were added to their diet. If the maternal diet did contain sugars during pregnancy and lactation, then the offsprings' teeth were caries-susceptible when sugar was added to their diet.

Calcification begins at the fifth month of fetal life and ends at about the third year in the deciduous teeth. For the permanent teeth it begins at the third to fourth month after birth and is completed after the eighteenth year. For this reason the deficiency diseases—such as scurvy, rickets, fibrocystic disease of the pancreas, celiac disease and the various infections of infancy and early childhood—may produce morphologic changes in the teeth making them more susceptible to caries.

It has been pointed out by Boyd[10] that dental caries can be controlled by diet but in his series all carbohydrates were given at mealtime and not between meals.

Just what influence heredity plays upon susceptibility to dental caries is not understood. If

one does not have dental caries, or has a very low susceptibility, prophylactic measures are not necessary. It is believed that the endocrine glands play no part in caries susceptibility.

Studies of the saliva of individuals who are nonsusceptible to dental caries have shown it to contain some protective substance. Also, when saliva is present in large amounts, it offers a natural washing of the teeth. Saliva is normally alkaline and it is highly buffered. Alkaline ash is contained in many fresh fruits and green vegetables and increases this buffering action. This action of saliva may explain why one person has rampant caries and his neighbor does not.

CONTROL AND PREVENTION

1. Repair—The only sure way of treating a cavity that has already formed is by restoration of the damaged structure. If the restoration is complete, there is reasonable assurance that the lesion may not progress. Restoration is the first step in caries control; this is the obligation of the dentist. Parents, physicians and dentists should be made to realize that deciduous or baby teeth require the same attention as the permanent teeth. When decayed they may produce pain, abnormal mastication, poor eating habits, gum boils and, when lost prematurely, malocclusion. Every tooth has a place and function in the mouth. The baby teeth serve the same function as the permanent teeth. They serve as functional units until the facial bones develop and the permanent teeth erupt in their proper place. Nature has provided an eruptive pattern. Each erupting tooth is dependent upon its neighbor for its position. This is also influenced by the muscular pressure of the lips, cheeks and tongue. Premature loss of a tooth may cripple the function of the mouth.

All forms of malocclusion should be under a dentist's observation.

2. Early education of the parents and children to the awareness and importance of oral health—This should have the same importance as other prophylactic therapy. If the parents are conscious of oral health they will accept more readily the responsibility of teaching it to their children. They should be advised to take the children to the dentist shortly after the second year for repair, prophylactic care and advice. This will instill confidence and reduce fear. Many good habits can be formed by the age of 5 or 6 years. Accurate advice can only be given if the physician himself is familiar with accepted knowledge of such care.

3. Children should be sent to an understanding and interested dentist. If the dentist realizes that he has the interest and help of the physician, much more cooperation will result.

4. A well-balanced daily diet with vitamins and sufficient rough foods is important. A large part of the necessary carbohydrates will thus be obtained without the necessity for a large sugar intake. Added raw fruits include not only sugars, but vitamins and minerals as well. What influence the maternal diet has on the morphology and susceptibility of the child's teeth has not been fully established yet.

5. Reduce the consumption of fermentable sugars, especially between meals and at bedtime. Sugar provides carbohydrates and nothing else. Jams, jellies and marmalades have, in addition, only a very small amount of protein. Bread, potatoes, milk, eggs, meats, fish, vegetables and fresh fruits provide not only carbohydrate but also protein, fat, calcium, phosphorus, iron, acid or base and vitamins A, B and C. In contrast, consider the following sugars consumed between meals by children: cola drinks and ginger ale (6 ounce bottle), 4 1/3 teaspoonsful sugar; 5-cent candy bar, 7 teaspoonsful sugar; chewing gum (one stick), 1/2 teaspoonful sugar; one tablespoonful thick chocolate sauce, 4 1/2 teaspoonsful sugar; 1 tablespoonful jelly, jam or marmalade, 2 1/2 to 3 teaspoonsful sugar; one serving of ice cream, 5 to 6 teaspoonsful sugar; cookies and cakes (depending on the amount and type), 1 to 15 teaspoonsful sugar. The other pops and frozen confections follow the same pattern.

6. Brushing the teeth—The old dictum that a scrubbed tooth does not decay has sold many toothbrushes but has had little effect on the dental caries rate. Brushing the teeth morning and night has become a ritual; so much so, in fact, that the equally important brushing after meals is difficult to establish as a habit. Brushing the teeth has three functions—stimulation of the gums, removal of food particles from all tooth surfaces and cleansing. Recently a fourth function has been suggested—the possible reduction or inhibition of acid-forming bacterial growth.

BRUSHING of the teeth should start before the eighteenth month of age. At that time usually a pleasurable habit can be started. It should be done after each feeding. A soft toothbrush

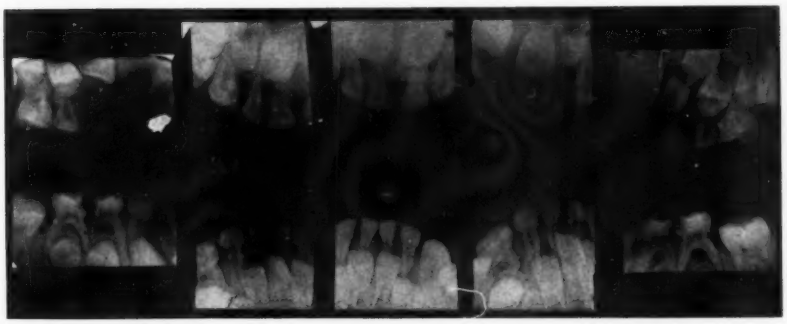

FIGURE 3. Dental film of the mouth showing nine large cavities of deciduous teeth.

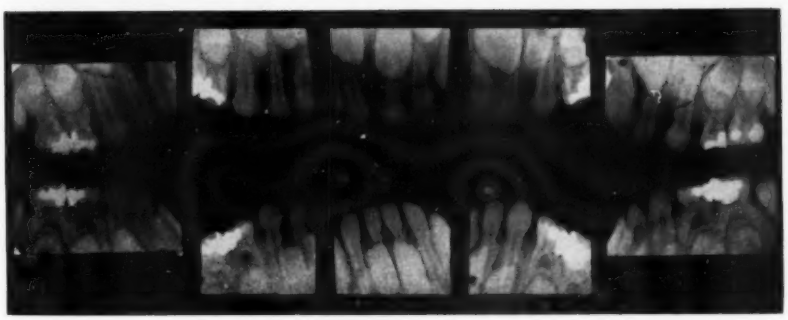

FIGURE 4. The same patient as in Figure 3 with nine filled deciduous tooth cavities.

and a pleasant tasting dentifrice, preferably a paste or watery solution, is best because there is less chance of harmful aspiration.

After the child has become accustomed to the tooth cleansing, the correct procedure for use of the toothbrush should be started. At first use a soft brush, small enough to fit the mouth. Later, a larger and stiffer brush should be used. The mouth is first rinsed thoroughly with water. Then, with the dentifrice, the bristles are placed firmly against the gingival dental margin and the brush rotated with a sweeping motion toward the occlusal surfaces. This will stimulate the gums as well as cleanse the buccal and interproximal surfaces because the bristles will be forced between the teeth. There should be five to six strokes over all areas until all the teeth have been included. Then a lateral motion on the occlusal surfaces will finish the job. The establishment of such a routine in early infancy should be attempted. Brushing at least after each meal and after the mid-meal consumption of food or sweets, if possible, is recommended.

7. *Rinsing the mouth*—As stated previously, fermentable sugars reduce the *p*H on the tooth surface from 7.0 to 5.0 within a few minutes after contact. During a meal, the increased amount of saliva and the intake of foods and fluids tend to dilute the substrate formed and aid in washing it away from the teeth. If brushing is not possible after the meal, rinsing the mouth with water is a good substitute. I have thought how much more logical it would be to use the finger bowl to rinse the mouth rather than to daintily wash the fingers. It would be a beneficial shock to society to make this change.

Rinsing of the mouth with water after taking between-meal foods, confections and drinks will at least partially wash away the substrate and the sticky high sugar foods. This should help if the present theories of sugar and bacterial initiation of caries are valid.

8. *Dentifrices*—We are being bombarded with advertising of dentifrices for which extravagant claims are made. The big seller at the present time is one that contains the ammonium ion.

The ammonium ion dentifrice was developed by Kesel[11] and others at the University of Illinois Dental School. It contains 5 per cent dibasic ammonium phosphate and 3 per cent urea. The theory behind the use of such a preparation stems from the finding of an inhibitory substance in the saliva of caries-resistant individuals. This substance is an ammonium com-

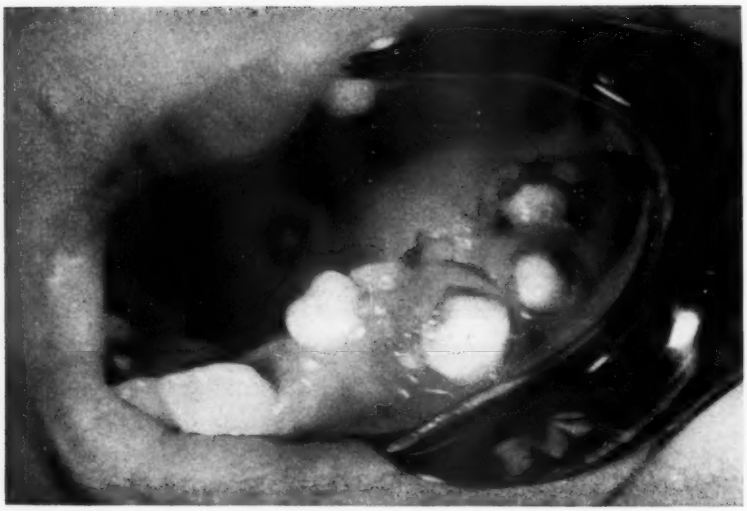

FIGURE 5. Severe dental caries and apical abscesses resulting in infection and irritation of gums and displaced eruption of permanent teeth.

FIGURE 6. Complete upper and lower dentures in a four-year-old boy. All deciduous teeth were carious or abscessed due to lack of dental care.

pound that does not neutralize but has a specific inhibitory effect on the growth of the acidogenic *L. acidophilus*. It is felt that the acidogenic organisms in the mouth are balanced by an equally important group of organisms that produce ammonium.

Ammonium ion dentifrices have been demonstrated to be effective in the reduction of oral bacteria but have not as yet been sufficiently proved to reduce the incidence of caries. Observations should soon be available to clarify this point. From our present knowledge these preparations are at least as good as any other dentifrice.

Reports on penicillin and chlorophyll dentifrices have not been sufficient to evaluate.

9. *Fluorides*—Systemic and topical applications of fluorides are being used to prevent caries. They act by developing a surface enamel that is more resistant to dissolution by acid.

Systemic fluorides—It has been reported that the use of a soluble fluoride, 1.0 part per million, in public drinking water will reduce the incidence of caries by as much as 60 per cent. To be of optimal value it must be used before the eighth year of age, during calcification and before the enamel is completely formed. Large scale testing of this procedure over a long enough period of observation should be available soon. When too much fluoride is available, it may be toxic.

Local application—Topical application to the teeth of a 2 per cent solution of sodium fluoride has been reported to be of value in caries prevention. Some studies have shown as much as 40 per cent reduction in the caries rate. To be effective it must be done by a dentist after a thorough prophylaxis. The teeth must be thoroughly dried and the fluoride allowed to remain on for four minutes. Four such treatments are given in a two to three-week period. It is recommended by the American Dental Association and some state health departments.

Fluorides in lozenges, tablets, wafers, dentifrices and milk, even if mixed with vitamin D and calcium, have been shown to be of no value. An insoluble calcium fluoride is formed which is not absorbed.

Added calcium and vitamin D are of no help if diet and vitamin intake have been adequate.

Research is in progress for some substance that will interfere with the reduction of sugar to acids. This would be the most economical way to prevent caries initiation by fermentable sugars. One study has reported the use of vitamin K in this capacity.

It is important for the physician to know and recognize when children are getting acceptable dental care. This involves a knowledge of the basic principles of oral hygiene and the accepted theories on caries initiation. It will mean a mutual understanding of the problem of pediatric dentistry by both the physician and the dentist. They should be in accord in the modern and most up-to-date precepts of oral health.

There should be more education of physicians for a better understanding of the problems of oral health, and medical students should receive some instruction in medical school on this subject. More articles should appear in the general medical literature on the principles, new theories and advances being made in oral health.

Dentists should be invited to participate in medical programs of local hospitals and county, state, national and special society meetings.

Some reading should be done by all physicians on oral health. The "Michigan Work Shop"[12] and "Sugar and Dental Caries"[13] can be recommended. Every doctor's library should contain a book on dentistry for children.

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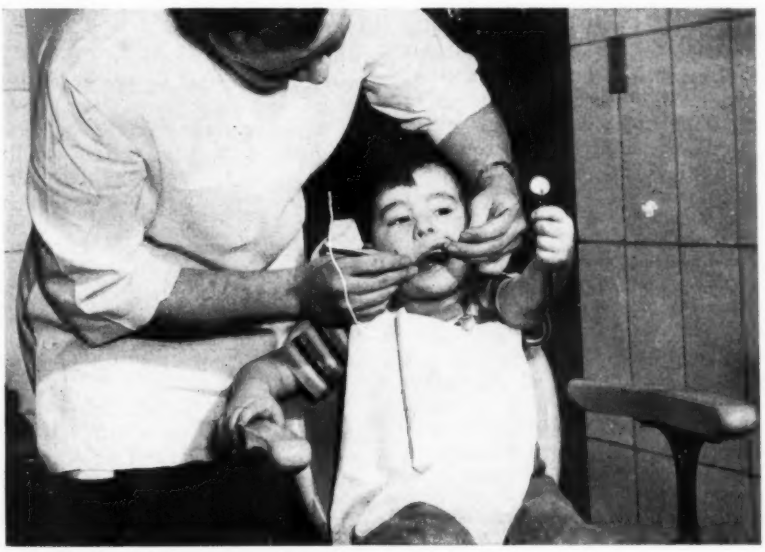

A VISIT TO THE DENTIST

(Photograph by Rus Arnold)

Case Report

Complications and Surgical Indications in Chronic Ulcerative Colitis

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Chronic idiopathic ulcerative colitis with its characteristic exacerbations is a serious disease. Whereas about 80 per cent of the cases can be kept under control with a strict medical regimen, the remaining 20 per cent frequently present themselves to the internist and the surgeon as a real problem. No longer should one wait until the victim of this disease is a debilitated, undernourished cripple with imminent lethal outcome before resorting to surgical intervention.

The indications for operation may at times be difficult to define but it is accepted by most that surgery is indicated (1) when efficient medical treatment has failed to produce cure or relief (intractable or incapacitated); (2) in the presence of complications—perforation, obstruction, massive hemorrhage, abscesses and fistulas as well as polyps—or possible malignancy. An interesting complication is a form of severe arthritis which usually does not subside until complete colectomy has been done; then as a rule complete disappearance of arthritis ensues.

No specific treatment has as yet been devised although some benefit has been obtained from sulfaguanidine, SULFATHALADINE, SULFASUXIDINE, antibiotics, etc. No doubt as an adjunct to surgery these drugs and antibiotics have real value.

The case to be reported is one of 3 severe cases treated by the author recently in which serious complications occurred, constituting an emergency. The proper surgical handling of these complications can tax the surgeon's ingenuity. Lahey[1] (1948) reported a case of massive hemorrhage complicating chronic ulcerative colitis where ileostomy was ineffective in controlling the hemorrhage. Although not done previously by him, a complete colectomy was done with recovery. On the basis of this experience, Lahey has reduced his stages for colectomy from four to two.

The present case presented not only massive hemorrhage, but incipient perforation of the cecum (disclosed at operation) and obstruction, and it was thought that the reporting of such a case might prove interesting and instructive.

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CASE REPORT

The patient was a 26-year-old white male married student. His illness began about June, 1945, while he was in the Army, when he noted bloody stools. He was told he had hemorrhoids and was treated with suppositories. Intermittent bloody rectal discharge continued. He was discharged from the Army early in 1946. Two months afterwards he began to have abdominal cramps. His stools were still well formed and bleeding was minimal. He entered a civilian hospital where a diagnosis of ulcerative colitis was made. He was placed on a medical regimen and his condition improved, with only occasional bloody stools. In March, 1947, he was readmitted to a civilian hospital because of frequent loose bowel movements with bleeding and abdominal cramps. He had been losing weight constantly.

In June, 1947, he was admitted to the Naval Hospital where under a strict medical regimen he began to gain weight and the stools became formed and bleeding ceased. He remained fairly well until October, 1948, when he again began to have frequent stools and noted loss of weight. This attack continued intermittently until about three weeks before admission to the medical ward of the Veterans Administration Hospital, Lebanon, Pennsyl-

vania on March 1, 1949, at which time in addition to his bloody stools he had chills and fever. Weight on admission was 97 pounds.

Physical examination revealed a young white adult, moderately emaciated with a pasty complexion, complaining of abdominal discomfort. There was some generalized rigidity of the abdominal wall with some superficial tenderness, but no masses. No anal fistulas or abscesses were present. The remainder of the physical examination was negative.

Laboratory data—Red blood cells numbered 3,960,000. Hemoglobin measured 12.3 gm. Total protein was 6.51—albumin 3.78, globulin 2.73. Urinalysis revealed 1+ albumin. Feces were positive for blood. The blood chloride was 561 mg. per 100 cc.

FIGURE 1. Flat plate of abdomen April 19, 1949; evidence of large bowel obstruction revealed by gas-filled colon. Of unusual significance is the outlined area representing the tremendously dilated cecum filled with blood.

THE patient was put on a low residue, high vitamin diet and given blood transfusions. Proctoscopic examination was difficult because of the extreme pain, but the area that could be examined for about 3 to 4 inches revealed ulcerations and pseudopolyps, corroborating the diagnosis of proctitis and ulcerative colitis. Report of a barium enema done shortly before in another hospital revealed involvement of the entire colon to the hepatic flexure. Additional therapy with sulfathaladine and penicillin brought about only moderate improvement. The patient continued to have abdominal cramps and became markedly depressed mentally. At this time the surgeon recommended early surgical intervention.

On April 19, the patient began to have severe abdominal cramps. The lower abdomen was somewhat distended and the author was asked to see the patient again in consultation. The patient had a fairly large bloody discharge from the rectum. Examination at this time revealed an acutely ill patient, although not in shock, with slight tenderness of the abdomen, moderate rigidity, and some distention. A transfusion was immediately ordered and the patient was observed for a few hours.

About 10 hours later examination revealed the patient to be in much more distress, with the lower abdomen distended to about the umbilicus, tender and rigid. The picture seemed to be one of massive intraluminal hemorrhage since no further blood was passed by stool. In addition, obstruction and possible perforation were considered. A flat plate of the abdomen taken earlier in the day was reported by the radiologist as revealing "some moderate distention of the large bowel on the left side and a tremendously distended bowel in the pelvis showing a mottling which was interpreted as feces" (Figure 1).

On April 20, 1949, the patient was operated on under ether and nitrous oxide anesthesia. Contemplating an ileostomy, a McBurney incision was made, but when the peritoneum was opened a large dense dark mass covered by a thin membrane resembling thinned out bowel was revealed. Moderate exploration was made and the mass was found to occupy the entire right lower quadrant, the pelvis, and left lower quadrant. The incision was extended mesially to the rectus muscle and upward to gain better exposure. Attempts to identify bowel structure were difficult because of the huge mass.

It was decided to deflate the mass by suction with a #20 needle; some gas was evacuated, and a trocar was then introduced. The mass was found to contain black blood clots. After some evacuation the mass was definitely recognized as cecum that had been extended to occupy the right lower quadrant, pelvis and left lower quadrant. Lateral to the trocar opening a minor perforation, partially sealed, was found. The terminal ileum was identified and this too was somewhat distended with blood. Due to the patient's very precarious condition, and to the fact that contamination from the perforation was no doubt present, a loop ileostomy was not done; instead, the cecum was exteriorized

FIGURE 2. Patient about one week after ileostomy, and approximately three weeks following the original cecostomy.

including the appendix, and using a large mushroom catheter, a cecostomy was done after the peritoneum had been closed. The patient responded fairly well to transfusions and antibiotics. Hemorrhage was obviously under control for the time.

Ten days later the patient, in fairly good condition, was operated on under cyclopropane anesthesia through a lower right rectus incision and the terminal ileum was found to be edematous for about 6 to 7 inches from the cecum. A site was then selected well away from this involved terminal ileum for the permanent ileostomy. After the ileum was divided between clamps the proximal end which was to be the permanent ileostomy was brought out in the lower part of the incision (Figure 2).

The ileostomy drained well and the patient's general condition improved. A Miller-Abbott tube which had been inserted prior to the ileostomy was removed on the fourth day. The abdomen was flat and the patient began taking nourishment by mouth and was permitted out of bed. The skin

FIGURE 3. Illustration of another patient with ulcerative colitis to show the Koenig-Rutzen bag in place. This patient had his hemi-colectomy about four weeks before this photograph was made.

FIGURE 4. This photograph represents the patient on the day of discharge 20 days after his abdominoperineal or final stage of his colectomy. One can see his well established ileostomy stoma as well as the nutritional improvement he has made.

surrounding the ileostomy as well as that surrounding the exteriorized cecum was kept in pretty good condition with kaolin and tannic acid. On May 31, the patient was given a Koenig-Rutzen bag (Figure 3) and was generally in very good condition. His weight at this time was about 121 pounds The patient was discharged to be readmitted for further surgery on July 7.

At this admission the patient weighed 137 pounds and except for the discomfort of the now small exteriorized cecum and occasional bloody discharge from the rectum he stated that he felt fine. For personal reasons he was permitted to leave the hospital and return on July 25 for his first stage colectomy, which was done on August 8. The right colon and about one-half the transverse colon were removed at this operation. The stoma of the distal half of the colon was exteriorized in the left upper quadrant through a stab wound. The patient responded very well. His ileostomy continued to function well and he was discharged on August 19, to be readmitted in three months for his final stage colectomy. Blood chemistry at this time was within normal limits.

He was readmitted October 30, 1949, for his final stage colectomy. At this time the patient weighed 140 pounds and his blood count was: red blood cells, 4,490,000; hemoglobin, 14.35; total protein 7.53—albumin 4.21, globulin 3.32.

The patient was prepared for his final stage abdominoperineal colectomy with preoperative penicillin and streptomycin; sulfathaladine solution was used for irrigating the remaining portion of the colon using a small rubber tube inserted into the anus. The last stage abdominoperineal colectomy was done on November 3. The postoperative course was uneventful. Minute attention was given to fluid balance as well as protein levels Penicillin, streptomycin and blood transfusions were given postoperatively. The patient began to take oral nourishment on the third day, at which time he was having ileostomy movements. The Levin tube was removed on the third day, and the patient was permitted out of bed. A small Penrose drain that was used in the perineal incision was removed and the wound was clean except for a very mild dark bloody drainage. He was discharged on November 23 in very good condition and his weight at this time was 137 pounds (Figure 4).

CONCLUSION

A THOROUGH and intensive preoperative preparation when time permits—this is always possible except in grave emergency—is most important. The patient should be prepared psychologically and with the help of a psychiatrist if available. Transfusions of whole blood, the use of vitamins, antibiotics and nonabsorbable sulfonamide drugs are

all important. Hypoproteinemia can play a very important role in the postoperative course and should be corrected, wherever possible, prior to operation. The free use of blood and proteolysates, both orally and parenterally, is emphasized. The introduction of a Miller-Abbott tube preoperatively, while not considered necessary by all surgeons, can be of great benefit during the immediate postoperative period.

As intimated elsewhere in this paper, a total colectomy can now be done safely in two stages instead of four. The author has had no personal experience with re-establishment of bowel continuity, nor in anastomosis between the ileum and the sigmoid in surgery of ulcerative colitis. Reports from other clinics where this procedure has been used are not too encouraging. In the case reported, the anal and rectal involvement precluded the saving of this portion of the bowel.

The use of blood and vitamins in the postoperative period cannot be stressed too strongly. The danger of a hypochloremia during the period after ileostomy when the physiology of the bowel is undergoing adjustment, necessitates frequent determinations of blood chemistry and correction by proper therapy. The care of the skin is greatly facilitated by the early use of the Koenig-Rutzen bag. Prior to its application many protective preparations are used, e.g., tincture of benzoin, tannic acid, kaolin, aluminum paste, etc.

An excellent presentation on this subject was made by Ferguson and Welty[2] in 1947.

The plea is repeated for early surgical treatment with subtotal or total colectomy in this very destructive disease so that these patients can again become useful members of society.

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BLACKWELL AWARD RECIPIENTS

BLACKWELL AWARDS, commemorating the anniversary of the first American woman physician and honoring achievements of women in medicine, were presented by the New York Infirmary to the five prominent women doctors shown here. They are, left to right: Dr. Marion E. Manter, director of internal medicine, New York Infirmary; Dr. Louise Pearce, pathologist and president of the Woman's Medical College of Pennsylvania; Dr. Edith Mass Lincoln, physician in charge of children's chest service at Bellevue; Dr. Sara Murray Jordan, leader in internal medicine and director of gastroenterology, Lahey Clinic, Boston, and Dr. Wilhelmina A. Ragland, director of obstetrics at New York Infirmary.

(Wide World)

GENERAL PRACTICE *Review*

To provide the general practitioner, as well as the specialist, with concise, readily available information on the latest, proved methods of treatment of conditions commonly encountered in an average practice, POSTGRADUATE MEDICINE *offers this department to its readers. It is not, of course, intended to present these discussions as the only acceptable therapeutic procedures to be used, but rather to offer simple regimens and recommendations based on the extensive experience of the physicians who prepared these summaries.*

Encephalitis

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A SHORT discussion of encephalitis is a difficult task since it must perforce leave much unsaid and must contain statements which need qualification, such qualifications being impossible in the space allowed. Then too our knowledge is incomplete, as in so many other problems, and there is difference of opinion. The very term itself—*encephalitis*—is used for involvements of the brain other than inflammatory ones. We shall confine ourselves to a consideration of the group which is generally accepted as deserving the term although, of course, even then there will be differences of opinion. One of the unresolved questions is: is the brain involvement in a specific infection the result of direct invasion by the organism or is it an allergic response?

GILPIN

There are several primary encephalitides in which a virus has been isolated. These include the St. Louis variety, the Japanese B and Australian X types, the spring-summer or Russian, the equine which includes the western and the eastern, and rabies. No organism has been isolated in the epidemic encephalitis group, formerly known as encephalitis lethargica. Of varied etiology are the many others which include toxoplasmosis, vaccinia, poliomyelitis, louping ill, pertussis, herpes simplex and zoster, varicella, mumps, measles, German measles, typhus fever, antirabic inoculations, virus pneumonia, other pneumonias, infectious hepatitis, infectious mononucleosis, influenza, rheumatic fever, undulant fever and typhoid fever.

The clinical manifestations of any of the above may be quite similar one to the other and indistinguishable except by bacteriologic and serologic means. In general the onset is apt to be fairly acute. There is fever, in some types mild and in some much higher. Mental changes are nearly always evident, ranging from irritability, insomnia, confusion to stupor or coma. In many there are pupillary and extra-ocular muscle changes. Disturbances of motility are frequent, either a movement disorder such as choreiform jerks or, on the other hand, paralysis. Usually present too are the signs of disseminated involvement of the nervous system. Most cases have meningeal signs present to a mild degree. Finally, in many instances the patient is left with some persisting sign or signs of the acute illness.

TYPES—FURTHER DESCRIPTION

Certain of the groups call for comment over and above the nonspecific description given in the last paragraph. The so-called epidemic encephalitis had become relatively rare. In the last few years however it seems to have been on the increase. Great caution must be exercised in making the diagnosis since so often it is incorrectly applied to some other disease. In spite of its seeming rarity in the acute

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stage there are far too many sufferers in the chronic stages who require care, particularly the parkinsonism cases.

It has been established[1] that the viruses of the St. Louis and the Japanese B types of encephalitis are transmitted by mosquitoes and that domestic fowl are the main reservoir for the infection of the mosquitoes. The virus of western equine encephalomyelitis is also transmitted by mosquitoes. Clinically these are indistinguishable one from the other and from the epidemic form. Ocular paralyses are uncommon in the St. Louis and Japanese types.[2] The pleocytosis found in the spinal fluid of the equine encephalomyelitis case is predominantly polymorphonuclear in type.[3]

The only way in which type can be determined and diagnosis made is by demonstration of neutralizing or complement fixing antibodies. In the Russian type, paralysis of the shoulder girdle muscles is common.[1] Rabies encephalomyelitis is most tragically seen when it occurs following the Pasteur treatment. The complication is now a rare one but sufficiently appalling to require that a wait of 14 days should be required to see whether the dog is rabid before giving the treatment. Then too the treatment should not be considered unless a bite with breaking of the skin has occurred.

Toxoplasmic encephalomyelitis is usually a postmortem diagnosis. It can be suspected in a child however when convulsions, hydrocephalus, cerebral calcifications, increased cells in the spinal fluid and chorioretinitis are found together. Laboratory serology confirmation is necessary. The occurrence of encephalitis after vaccination against smallpox is not frequent but can be kept at a minimum by vaccinating sometime in the first year when the incidence of the complication is lowest.

Measles is accompanied by encephalitis in about one case in a thousand.[4] It has its onset about two to six days after the onset of the rash. The prognosis is guarded and residual symptoms such as mental defect, paralysis and ataxia are less likely in patients with a severe acute phase of the illness.

Whooping cough is not infrequently complicated by encephalitis;[5] in fact, it, more than any other infectious disease, may be so accompanied. It occurs more often in younger children and it is more frequent in the more severe cases with more severe cough. It appears generally about the third week of the disease. The prognosis is grave. Convulsions are the most frequent symptom. Mental and motor sequelae are very common (50 per cent).

Mumps is accompanied by invasion of the nervous system in at least a third of the patients.[6] The clinical evidence of this is not so often clear except in the spinal fluid examinations. The nervous system involvement may precede the appearance of the parotid swelling[7] and on occasion may even be the only manifestation of the presence of the virus. Prognosis is good.

It seems worthy of emphasis that encephalitis does occur as the result of the virus of herpes simplex invading the nervous system. German measles may even be accompanied by neurologic signs and symptoms occasionally, as may infectious hepatitis and infectious mononucleosis. It is well recognized that rheumatic fever may exhibit a cerebral phase, namely, chorea, but it is not so well known that other symptoms may develop as well.[8] These may include delusions, delirium, disorientation, masked facies, one-sided weakness and ocular palsies. Salicylate therapy does not influence this.

The diagnosis of encephalitis, in the first place, requires that the possibility be kept in mind and this must be balanced against the relative infrequency of the condition compared to other diseases which may be confused with it. Poliomyelitis will be recognized by the paralysis. Tuberculosis of the meninges produces typical chemical changes in the spinal fluid—a very low chloride approaching 550 mg. per cent. Syphilis of the nervous system is accompanied by the serologic evidence of its presence. Brain tumor and a vascular lesion of the encephalon may offer difficulty occasionally but time will usually resolve the problem.

MANAGEMENT OF ACUTE STAGE

The management of the acute stage of encephalitis is unfortunately confined to symptomatic measures. Some of the newer antibiotics have been tried with great hopes but as yet no really accepted results have been forthcoming. Two suggestive reports have appeared which may point a way however. The one was concerned with the effect of ether anesthesia on experimental western equine encephalomyelitis.[9] The attempt was to bring about a reversible change in metabolism of the host cell sufficient in degree to destroy the virus without causing permanent injury to the host cell. The second report[10] was concerned with the inhibition of mumps virus multiplication by the capsular polysaccharide of type B Friedlander bacillus. Vaccines are available for some types of encephalitis. Mosquito control is essential.

POSTENCEPHALITIC PARKINSONISM

No discussion of encephalitis should fail to consider at least briefly the treatment of the most frequently encountered sequel—parkinsonism. The picture in that disease is so well known as to warrant no repetition here. The treatment is not so universally understood.

The primary symptoms, rigidity and tremor, present the problem. Nearly all drugs are more efficacious in relieving the former than the latter. They include atropine, belladonna and stramonium of the older official preparations together with such

proprietary drugs as VINOBEL and RABELLON. Hyoscine or scopolamine, in general, has a more beneficial effect in controlling the tremor than the previously mentioned drugs do. In addition to those listed there are four newer preparations which deserve comment. First to be mentioned, because it seems to be the best of all available up to now, is ARTANE, a synthetic which has effects similar to atropine but with milder toxic reactions. The patient must be adjusted to the proper dosage by trial and error. The extremes vary between 1 mg. four, five or even six times per day and as much as 40 mg. in 24 hours. Many will require the more frequent administration, as often as every two to three hours, but some will be content with 3 doses per day. The tremor may not respond sufficiently to artane and hyoscine may have to be added, starting with 0.3 mg. per dose.

The others of the newer group include PANPARNIT, the antihistamines, particularly BENADRYL and MYANESIN or TOLSEROL. Panparnit can be administered in 12.5 mg. dosage units varying the frequency and amount according to the needs of the case. Toxic symptoms are more frequent and the beneficial effects less than with artane. However, in this group of patients there is great individual variation in response to the various drugs and in any given case any one preparation may be superior to all others. Then too after one has taken any of the drugs for a while, he obtains less and less benefit so that a change is necessary.

Benadryl adds something in some instances, possibly because of its psychologic and sedative effects. Tolserol or myanesin orally has little to recommend it.

Some patients will be helped by amphetamine sulfate 5 to 40 mg. per day. These are particularly those with a disorder of the sleep mechanism and those with oculogyric crises.

In addition to the drug therapy, physical therapy is often a very necessary and helpful adjunct. The psychologic aspect of the problem must not be forgotten. It requires much tact, encouragement and suggestion to keep these chronically ill patients going and they should be kept active as long as is even faintly possible.

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Treatment of Contact Dermatitis

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CONTACT dermatitis is an inflammatory reaction of the skin due to the external action of either physical, animal, vegetable, mineral or chemical substances. This inflammatory reaction may consist of erythema, edema, papulation, vesiculation, oozing or crusting, or any combination of these lesions. It is usually followed by desquamation, lichenification and pigmentation.

DOWNING

The first step in therapy is correct diagnosis and determination of the causative factor. A careful history and patch tests are valuable, for removal of the cause should prevent a continuation of the eruption. A classic reaction from exogenous factors such as poisonous plants consists of erythema, edema and vesiculation. Therapy consists of hot and cold wet dressings; then a soothing lotion and bland paste. It is essential that the treatment be continuous. Wet dressings should be changed every two hours for 24 to 48 hours. The lotion should then be applied every hour for 24 to 48 hours until the skin becomes dry and desquamating. Finally

the paste is applied and renewed several times a day. At the end of 24 hours these applications should be removed with bland oil.

During the acute stage, washing with soap and water and friction due to cleansing should be carefully avoided. If a superficial infection appears, wet dressings of boric acid solution or potassium permanganate 1:5000 may be used for one hour daily before reapplying the lotion and paste. If the eruption becomes generalized, baths are valuable for cleansing and soothing the skin. Cornstarch, bran or a combination of oatmeal and sodium bicarbonate, the so-called colloid bath, should be used for this purpose.

Powders are valuable because they are cooling and drying, especially on red, inflamed unbroken skin. The mineral substances are best—talc, zinc oxide, calcium carbonate, magnesium carbonate and kaolin. A reddened healing area may be protected by borated talc. The incorporation of a small amount of oil is helpful, such as

Magnesium stearate	12
Liquid petrolatum	12
Talc	q.s. 120

Suspensions of powders—lotions—should be used on congested and oozing surfaces. Two useful and popular emulsions are calamine lotion U.S.P. XIII and neocalamine lotion N.F. VIII containing a colloidal clay called bentonite which helps to produce smooth emulsions. The addition of phenol, 1 to 2 per cent, and menthol, 0.25 to 0.5 per cent, relieves the pruritus. The following is an excellent formula for relief of severely itchy, vesicular areas:

Zinc sulfate	2
Phenol	2
Zinc oxide	15
Lime water	q.s. 250

This can be colored to match the patient's skin by adding neocalamine powder in proper proportions.

Solutions of tannic acid and ferric chloride and concentrated potassium permanganate are advocated and useful in the treatment of the dermatitides resulting from contact with poisonous plants, but the patient is apt to forget the former suffering and discomfort and condemn the physician who has discharged him with a stained or pigmented skin. They never should be used on areas of the skin exposed to sunlight, because it accentuates and prolongs the disfigurement.

When the acute inflammation has subsided, pastes or emulsions are indicated. An excellent watery paste is:

Zinc oxide	30
Talc	30
Glycerin	30
Water	30

The official paste of zinc oxide is popular, but I prefer

Zinc oxide	2
Hydrolyzed cornstarch	13
Petrolatum	15

or the following:

Solution of aluminum acetate	10
Lanolin	20
Zinc oxide paste	30

Pastes should be used after an antipruritic lotion. However, an emulsion or liniment might replace both. Calamine liniment N.F. VIII or the cuticolored neocalamine liniment N.F. VIII satisfies all requirements. When an antipruritic is needed, phenol or menthol may be added in reduced amounts to avoid irritation due to the prolonged action in the oil. If there is any superficial infection, the following formula is indicated:

Menthol	0.5
Phenol	1.0
Ichthammol	7.5
Zinc oxide	30.0
Olive oil	
Lime water	aa ad 250

The use of ointments is the last step in the treatment of acute eruptions of dermatitis venenata. Having no evaporation properties they cause congestion and irritation if applied too soon. When properly used they are of extreme value. When the eruption becomes dry, crusted, desquamated, fissured or lichenified, a bland ointment such as one of the following may be indicated: white petrolatum, AQUAPHOR, rose water ointment, boric acid ointment, zinc oxide ointment, or the nongreaseless hydrophilic ointment base, U.S.P. Pastes and ointments should never be applied directly to the skin, but rather on a piece of soft linen. Too vigorous removal of these applications will delay healing; they should be sponged off gently with sterile oil. Three per cent VIOFORM ointment is of value if there is superficial infection. A very useful, soothing ointment is calamine ointment N.F.:

Prepared calamine	17
Yellow wax	4
Wool fat	4
Petrolatum	75

The addition of a small amount of neocalamine will make this ointment skin color.

Tars should never be used in the treatment of the early stages of a contact dermatitis, but they are of great value in persistent pruritic patches of weeks' duration. White's original formula is of great benefit:

Crude coal tar	2
Zinc oxide	2
Cornstarch	15
Petrolatum	15

The newly introduced group of so-called antihistaminic ointments are of value in certain selected cases. Five per cent THEPHORIN ointment has been beneficial in a carefully selected group of persistent dermatitides. Two per cent PYRIBENZAMINE cream and 2 per cent HISTADYL cream are also of value in lichenified patches. However, one must be careful of the occasional sensitization reaction produced by these drugs. It is apparent that even the so-called antihistamines produce allergies in susceptible individuals.

Attempts at desensitization, despite the occasional report of success with plant oils, have not proved of value. They should be used cautiously, for they may cause severe reactions; this is true also as regards prophylaxis. Conflicting reports are published regarding even the value of *Rhus toxicodendron*. Injections of this antigen may be dangerous. Antibodies have not yet been demonstrated in cases of contact dermatitis, but more delicate methods may yet reveal their presence.

In the treatment of contact dermatitis the physician must consider the patient as an individual. There is a great emotional reaction to a severe outbreak of the skin. He or she must be reassured that the eruption is curable, that injudicious self-treatment, neighborly advice, exercise and indiscretion of diet will prolong the eruption. A careful, thorough examination and thoughtful advice with explicit directions as to the use of prescribed therapy will shorten the duration of the disease.

Treatment of Acute Cholecystitis

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COLE

ALTHOUGH the major intent of this presentation is to discuss the treatment of acute cholecystitis, I wish to summarize some of the data on pathogenesis and manifestations because certain of these features exert such an important role in the management of the disease. It is well known that stones are present in the gallbladder in 97 to 98 per cent of patients with acute cholecystitis. On most of these occasions a stone usually blocks the cystic duct temporarily, if not permanently. One can understand that this might lead to a condition requiring urgent care. It is not generally appreciated that acute cholecystitis may not be infectious in origin. The author is convinced that in a great many cases the inflammation starts out as chemical and infection becomes superimposed later. For example, in a recent study made by Lester,[1] the bile cultures in nongangrenous cholecystitis were positive in 28 per cent, whereas they were positive in 59 per cent of patients with gangrenous cholecystitis. It is obvious then that if infection is not present or is not virulent, considerable operative work can be carried out in the presence of acute cholecystitis without the danger of a spreading infection.

Manifestations of acute cholecystitis consisting of pain in the right upper quadrant, nausea and vomiting, tenderness over the gallbladder region and fever are fairly constant. Muscle spasm will be present in most cases depending somewhat upon the acuteness of the inflammation. The presence of jaundice is a complicating factor which must be considered in the type of treatment advised. On some occasions, the jaundice will be caused by stones in the common duct, but usually it is due to acute hepatitis, particularly when the jaundice is of relatively mild intensity. In a series of 109 patients with acute cholecystitis studied by Lester, 17 per cent were jaundiced but stones were found in only 4.6 per cent, although the common duct was opened in a few patients where dilation of the common duct was present without demonstrable stones.

It should be emphasized that when acute perforation develops, manifestations of generalized peritonitis appear quite rapidly. However, the amount of muscle spasm varies considerably and

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is dependent largely upon the concentration of bile escaping in the free peritoneal cavity, and the presence or absence of infection. In peritonitis following free perforation in the peritoneal cavity there may be, and in fact usually is, no acute localization in the right upper quadrant.

WHETHER OR NOT TO OPERATE

Before any decision is made as to the treatment indicated, it is obvious that a thorough search must be made for complicating diseases. Many complicating diseases may make an emergency operation undesirable.

During the past two decades there has been a gradual shift away from conservative treatment in acute cholecystitis to emergency operation. One of the reasons for adoption of this radical view by many surgeons is the fact that the operative mortality rate is about as low following cholecystectomy for acute cholecystitis as it is following cholecystectomy for chronic cholecystitis. Glenn[2] reported a mortality rate of 3.5 per cent in 170 patients operated upon for acute cholecystitis in the past four years. This rate is higher than in cholecystectomy following uncomplicated chronic cholecystitis, but is no higher than that following cholecystectomy for all types of chronic cholecystitis including stones in the common duct.

If the mortality rate is to be used as a means of making a decision, one would have to include at least some patients with stones in the common duct in the group of cholecystectomies for chronic cholecystitis, since stones were found in the common duct in 5.8 per cent of Glenn's series. Accordingly, it is therefore not entirely valid to use mortality rate as a factor in arriving at a decision as to whether or not emergency operation is justifiable, or indicated. It is very true that emergency cholecystectomy shortens the discomfort, disability and period of hospitalization. However, the surgeon should not allow these factors to exert a very important role in the decision as to type of definitive treatment since the most important feature in this disease actually is to save the patient's life. It must likewise be admitted that emergency operation will prevent perforation which obviously will result in a higher mortality rate when conservative treatment is adopted. It must be remembered that perforation is most likely to occur in patients who have had no previous attacks. This is particularly true of free perforation into the peritoneal cavity, since previous attacks are apt to result in so many adhesions that the perforation itself may become localized by omentum, intestinal loops, etc.

In fairness, it should be stated that many surgeons still favor conservative treatment of acute cholecystitis largely because they feel the mortality rate will be lower following this type of therapy. All surgeons would obviously operate on a patient if perforation were suspected. A great many surgeons adopting the conservative method of treatment would resort to emergency operation in a patient with high fever and acute localizing symptoms on the basis that perforation may develop even though it is not present at the time the patient is seen.

In the author's opinion no flat rule can be made as to whether conservative treatment or emergency operation is indicated in acute cholecystitis However, he is in full accord that in the majority of instances the operative treatment is justifiable. One of the dangers of a wholesale adoption of operation in acute cholecystitis is that there will be a very high incidence of error in diagnosis when the operation is performed shortly after admission of the patient to the hospital. The importance of this statement is emphasized by the fact that in a series of patients reported by Pennoyer,[3] of 59 patients with acute cholecystitis operated upon as emergencies, 54 per cent were actually diagnosed as having another disease. The author believes that in general, if a patient is seen within 48 hours after the onset of symptoms emergency operation is justifiable. If the symptoms are of longer duration than 48 hours we are inclined to adopt a conservative attitude on the basis that the inflammatory process has progressed rather widely and infection may have become superimposed. These two factors would obviously increase the mortality rate considerably.

If the conservative method of treatment is adopted certain precautions must be taken to prevent perforation while the patient is under medical care. Even though operation is performed very shortly after free perforation of the peritoneal cavity, the mortality rate will be high in this group. Accordingly, the author has adopted the following principles when conservative treatment is being utilized:

1. If the patient's fever increases, or does not drop to near normal within 24 hours after admission to the hospital and adequate care, gangrene of the gallbladder wall may be present as the explanation. Occasionally, empyema will be the explanation of the fever, but if this is present and gangrene not imminent, the local findings will be minimal. Accordingly, persistence or increase in fever is an indication for emergency operation.

2. If the local findings increase, or are permanent without regression, gangrene may be imminent. Obviously, muscle spasm will be an important factor in the local findings. The development of muscle spasm, particularly if it is progressive in extent, is a fairly good indication that perforation is threatening. Accordingly, increase in local findings constitutes an indication for emergency operation.

3. If pain increases in severity or fails to decrease, there may be indication that the inflam-

matory process is proceeding to complications such as gangrene or perforation, and operation will then be advisable.

Accordingly, any of the factors listed may be interpreted as indication for immediate operation largely on the basis that gangrene or perforation is imminent and should be prevented by operation. The type of operation to be performed is open to question, and the various factors in making that decision are discussed below in detail.

INDICATIONS FOR CHOLECYSTOSTOMY

In elderly patients, who obviously are poor risks because of cardiac lesions, extreme senility, etc., there may be strong indications to do a cholecystostomy instead of a cholecystectomy when operation is performed. On most occasions, cholecystostomy will be indicated if perforation or gangrene is present, particularly if peritonitis has developed. When the inflammation is so acute that the procedure of cholecystectomy would result in trauma and spread of infection, if present, cholecystostomy is usually the procedure of choice.

When in doubt as to whether cholecystectomy or cholecystostomy should be performed it is very helpful to aspirate the gallbladder and examine the fluid under the microscope. If the fluid contains a lot of granular material including cholesterol precipitate, and no bacteria is found, one is fairly safe in proceeding with cholecystectomy, assuming that the operative risk is good. On the other hand, if the smear is loaded with pus cells and numerous bacteria, cholecystostomy may be the procedure of choice, particularly if the organisms are streptococci. It is true, however, that we do take a few chances under such circumstances, particularly since we now have fairly effective antibiotics. However, it is very dangerous to assume that infection can be controlled completely by antibiotics.

If the common duct is obstructed in the presence of acute cholecystitis it is usually desirable to open the common duct, remove the stone and perform a cholecystostomy, since removing the gallbladder in addition to a choledochotomy might be too big a load for the patient's reserve.

INDICATIONS FOR CHOLECYSTECTOMY

In all patients whose physical condition permits and in whom the amount of inflammation is not maximal, cholecystectomy may be considered, particularly if it appears that the gallbladder can be removed without a prolonged and rather difficult dissection. As previously stated, examination of a smear taken from the gallbladder contents will be helpful in deciding whether or not cholecystectomy is permissible. One must bear in mind that cholecystostomy often does not cure the patient. For example, in a series of 149 secondary operations (primarily cholecystectomies) on the biliary tract, reported by Foss,[4] 100 had cholecystostomy as the primary operation. Since the ratio of cholecystostomy to cholecystectomy is much less than this, it is obvious that cholecystostomy will be followed by recurrence of symptoms in a great many instances. Moreover, the mortality rate of 8 per cent in his secondary operations on the biliary tract is a point favoring cholecystectomy rather than cholecystostomy at the time of the original operation for acute cholecystitis.

After reviewing the data on results reported by numerous surgeons following cholecystostomy, and my own experiences, it is obvious that 30 to 40 per cent of patients having cholecystostomy for acute cholecystitis will return within five years with serious symptoms of gallbladder disease.

INDICATIONS FOR CHOLEDOCHOSTOMY

To a great extent one utilizes the prerequisites of choledochostomy in chronic biliary disease when considering opening the common duct in acute cholecystitis. In other words, if the patient is jaundiced and a stone is palpable in the common duct it should be opened and the obstruction removed. The only exception is a patient who is a very poor risk, and whose cystic duct is patent. Under these circumstances, cholecystostomy would relieve the obstruction. However, it must be remembered that in acute cholecystitis the cystic duct is usually blocked. In acute cholecystitis any operative treatment which does not remove an obstruction of the common duct is poor therapy; failure to do so may result in a fatality. In general, any patient having jaundice at the time of operation for acute cholecystitis must be considered as a candidate for choledochostomy. However, as has been mentioned previously, less than half the patients with jaundice will have stones in the common duct. Glenn opened the common duct of 8.75 per cent of patients on whom he operated for acute cholecystitis, and found stones in 66 per cent of this group. However, he did not do choledochostomies in all patients with jaundice. Accordingly, one should utilize color of the stool, size of the common duct and thickness of its wall in determining whether or not the duct should be opened. If the patient is jaundiced and his physical condition not critical, a dilated common duct should be opened even though a stone cannot be palpated within it.

In acute cholecystitis, a previous history of jaundice associated with pain in the right upper quadrant is not an indication for opening the duct unless a stone is palpable within it. As stated, about half the patients with acute cholecystitis and jaundice will have hepatitis as the cause of jaundice. In such patients, choledochostomy is definitely an error, although by no means a fatal one.

PRE- AND POSTOPERATIVE CARE

Ordinarily pre- and postoperative care following operations on the biliary tract are not complicated. One of the important features in preoperative care is to assay fluid balance and correct dehydration and electrolytic balance. Dehydration and electrolytic loss are not pronounced unless the patient has been vomiting, or unless he has failed to eat or drink during the previous days. The amount of fluid, such as 5 per cent glucose with or without saline, is determined by the amount of vomiting or intake just preceding admittance to the hospital. Since symptoms are usually acute there rarely will be a hypoproteinemia unless a complicating disease exists. Rarely will a preoperative transfusion be necessary unless a complicating disease is present.

Postoperative gastric suction is indicated for a period of 48 hours. This is important since postoperative ileus following operation on the biliary tract (particularly cholecystectomy) is fairly severe and constant. The decompression will prevent distention and gas pains. Removing the tube in 48 hours usually allows the patient to begin eating and rapidly taking a soft diet.

RESULTS

The ultimate results in acute cholecystitis will depend on several factors including type of therapy instituted, the severity of the infection and physical condition of the patient. In a series of 697 patients treated by the operative method during the past 17 years, Glenn[2] reports a mortality rate of 2.7 per cent. The rate during the past four or five years (1946-1950) was no less than that for previous years; it actually was slightly higher. This indicates that improvements in our knowledge of physiology, chemotherapy, etc., gained during the past few years have exerted no benefit in results in acute cholecystitis, presumably because nutritional deficiencies and infection are not significant features of this disease. It is very significant that in Glenn's series the mortality rate in patients over 50 years of age was 4.9 per cent, compared to 1.4 per cent in those under 50 years of age (ratio of approximately 3.5 to 1).

The results of conservative treatment are well illustrated by Pennoyer's series, in which the mortality rate was 5 per cent in 241 cases. Of this number, the mortality rate in 208 cases which subsided was 2.5 per cent contrasted to a rate of 20 per cent in 34 patients who were operated upon because of perforation or threatening perforation.

The mortality rate of cholecystectomy cannot be compared to the rate for cholecystostomy from the standpoint of danger of the operation, because with few exceptions the former procedure will be chosen in patients with mild inflammation and optimum operability. Nevertheless, it should be emphasized that cholecystostomy is a much safer operation than cholecystectomy and should be used when operability is poor and inflammation intense.

To a great extent the mortality rate in any series will be strongly influenced by the number of free perforations into the peritoneal cavity, because the mortality rate is so high in this group, namely 60 to 75 per cent.

Symptomatic results will depend upon the number of cholecystectomies and cholecystostomies in a series. There is better than a 95 per cent probability that the patient will be asymptomatic following cholecystectomy for acute cholecystitis, providing no stones are left in the common duct. On the other hand, after studying data in the literature and reviewing his own experience, the author is convinced that no more than 30 per cent of patients with cholecystostomy will remain entirely asymptomatic if the patients are observed for at least 5 years after operation. Of the 70 per cent remaining, about half (35 per cent) will have such severe symptoms as to justify another operation, primarily cholecystectomy.

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Treatment of Mumps

EDWARD B. SHAW

SAN FRANCISCO

UNTIL comparatively recently the prevention and treatment of mumps was a matter of little importance. The infection was largely a nuisance disease which children were expected to acquire—and usually did. Most older textbooks give little attention to its treatment except for the use of obvious symptomatic measures.

SHAW

Because of the comparatively low communicability of mumps, this is the one childhood infection which is most frequently escaped in childhood, only to occur with more serious symptoms and a higher incidence of complications in adulthood. Of late, there has been less acceptance of the necessity for children to acquire the common minor infections. More careful protection of children against exposure, which in the case of mumps is perhaps mistaken, has resulted in this disease becoming more and more one of adult years.

The diagnosis of mumps in the usual form is easily established. Typical swelling of the salivary glands, especially of the parotid, with some overlying gelatinous edema, the frequent hemorrhagic injection of the stomas of Stenson's duct, the absence of purulent secretion from this duct and the accompanying leukopenia serve quickly to establish the diagnosis.

The disease seldom occurs in infancy although it occasionally does so. Throughout childhood mild symptoms of pain and tenderness are usual, severe pain and general prostration being less common; adults are frequently much more severely ill.

There are almost no serious effects from infection of the salivary glands and the principal significance of mumps results from involvement of other organs. Any of the so-called complications (i.e., infections apart from the salivary glands) may occur before, during or after the recognition of parotitis and may occur without any recognizable salivary gland involvement.

LABORATORY TESTS

Leukopenia is usually present although extra-salivary gland infections may cause leukocytosis. A newly available antigen for skin testing will demonstrate susceptibility to the mumps virus. Serologic tests (complement fixation and red cell agglutination inhibition) which are negative at the beginning of infection and become positive two or three weeks later effectively prove the specific etiology.

COMPLICATIONS

Orchitis is the traditional complication of mumps; this virtually never occurs in pre-adolescent males. In adult males it occurs in about 20 per cent of all cases, causing great swelling of one or both testicles with associated pain, tenderness, prostration and apprehension. Resultant atrophy of the affected testicle is said to occur in about 20 per cent of the testicles involved but because both testes are

rarely equally affected the incidence of actual sterility as a result is probably considerably less than 1 per cent of all cases in which orchitis occurs.

Oophoritis probably occurs in women with comparable frequency, causing low abdominal pain and tenderness, but it is less susceptible to accurate diagnosis. The distress caused by this complication either is less severe or the female exhibits more fortitude.

The virus is said to involve the female breasts although this must be extremely rare.

Pancreatitis may occur with varying degrees of upper abdominal pain, vomiting and prostration. This diagnosis may be relatively easy if parotitis precedes the symptoms. As in the other complications this may not be directly related to recognizable parotitis and the diagnosis may finally be established only by serologic tests. Serum amylase levels may rise to considerable heights in pancreatitis although this may occur also in uncomplicated parotitis.

Central nervous system infections are extremely common in patients with parotitis. These usually consist of evidence of meningitis with moderate to severe headache, prostration, vomiting and neck stiffness. As in other forms of meningitis, there is sometimes somnolence and lethargy but cranial and peripheral nerve palsies are extremely rare. The only fatality of which the writer has personal knowledge was due to pulmonary edema. The central nervous system form of the disease is usually termed mumps meningo-encephalitis, but the principal findings are those of meningeal inflammation with a minimum of encephalitic signs. There are many mild cases in which clinical evidences are equivocal. The diagnosis may be strongly suspected in benign forms of nonbacterial meningitis in which the spinal fluid cell count is elevated to 100 to 1000 with an increase in monocytes to 90 or 95 per cent and with normal chemical findings. The final diagnosis may be indicated by a history of exposure, by associated parotitis, and may be finally confirmed only by serologic tests.

Deafness is ascribed to mumps with sufficient frequency to suggest an actual causal relationship. This is probably related to mumps meningitis in which the resultant deafness is comparable to that which frequently used to complicate septic meningitis.

TREATMENT

Parotitic infection in childhood involving one or more of the salivary glands is almost invariably succeeded by lifelong immunity and is rarely accompanied by any serious sequelae. Accordingly, whether or not infection with mumps should be actually encouraged, nothing should be done during childhood to prevent the disease following exposure except in most unusual circumstances.

ACTIVE IMMUNIZATION

Methods for the prevention of mumps in childhood are largely inappropriate. In older age groups susceptibility may be determined by means of the skin test and active immunity can be stimulated by injections of the commercially available chick embryo culture virus. This modified virus is incapable of producing typical signs of the disease; it does induce active immunity although this immunity cannot be demonstrated by means of a subsequent intradermal test. The prolonged incubation period makes it possible, when desirable, to induce active immunity following known exposure.

TREATMENT OF SALIVARY GLAND INVOLVEMENT

The treatment of simple parotitis should be entirely symptomatic. Aspirin in combination with codeine may be used for relief of pain. Local applications of heat or cold (or the ancient device of applying camphorated oil!) are of little value. Oral hygiene should be promoted by the use of bland mouth washes such as bicarbonate of soda and water; many of the more highly flavored mouth washes will provoke some pain. The patient should be kept at rest in proportion to the amount of discomfort and prostration which is evident, although complications cannot unfailingly be prevented by complete bed rest.

ANTIBIOTICS

It is quite logical to expect that some one of the recently developed antibiotic agents would be effective against the large-sized virus of mumps except for the fact that no one of these has been shown to be effective clinically in the prevention of infection of the chick embryo, or in prevention or treatment of the disease in experimentally infected monkeys. Penicillin, aureomycin, CHLOROMYCETIN® and terramycin all seem to be ineffective in the inhibition or destruction of the virus. There are scattered reports which favor the use of aureomycin but this is experimentally ineffective and a recent report notes the development of mumps in a patient who was under treatment with this agent.

HUMAN SERUM PREPARATIONS

Serum from a patient recently convalescent from mumps contains appreciable amounts of antibodies against the virus but this serum is valueless in the treatment of active disease in the salivary glands or elsewhere. The use of convalescent serum immediately after exposure may somewhat reduce the incidence of the disease but is not completely dependable. Recommended dosage is 20 to 40 cc. given within seven days after exposure. There is some

prospect that gamma globulin prepared from convalescent serum may be available in the future and this should accomplish a comparable effect in smaller dosage. Variability in the infection rate makes it impossible precisely to evaluate serum prophylaxis but it is very much less effective than the similar use of convalescent serum in the prevention of measles. Many failures of protection occur and these are particularly annoying in the prevention of infection following hospital ward exposure.

PREVENTION OF COMPLICATIONS

Convalescent serum is frequently recommended for the prevention of complications following parotitis, especially orchitis in the adult male. The use of convalescent serum for this purpose is apparently attended with partial success although percentage-wise it seems never to be completely effective. A dose of 20 to 40 cc. is usually recommended with some promise that the probability of orchitis may be diminished. In a series reported by Gellis[1] the use of gamma globulin prepared from convalescent serum reduced the incidence of orchitis in adult males to 7 per cent compared to a control series treated with 50 cc. of pooled normal plasma in which the incidence of orchitis was 24.7 per cent. The latter figure agrees with the usual 20 to 25 per cent in which this complication may be expected. Obviously, however, this protection falls far short of the value of gamma globulin preparations in the prevention of measles.

Pooled adult plasma and gamma globulin obtained from normal plasma cannot be depended upon for any protective effect against mumps or in the prevention of complications.

TREATMENT OF COMPLICATIONS

Orchitis—As already indicated, the use of immune serum at any stage of the disease cannot be completely relied upon to prevent orchitis and once this complication has developed convalescent serum seems to be without value.

All of the antibiotic agents are without specific effect.

Estrogenic hormones have been employed without any convincing evidence of their value in well controlled studies.

To a large extent the extreme pain and subsequent atrophy of the testicle are caused by the great tension of fluid beneath the tunica albuginea in the inflamed testicle. According to Wesselhoeft[2] simple incision through the tunica albuginea early in the infection will relieve the pain and greatly improve the likelihood of recovery without atrophy. Apparently this may be accomplished by a simple stab wound through the anesthetized skin although more extensive incision is frequently employed.

Procaine block of the spermatic cord at the level of the external ring was proposed by Kulenkampff.[3] According to Smith[4] the theory of this treatment is that pain and swelling are related to neurovascular factors. The sympathetic nerves which pass into the testicle may be blocked by infiltration with 10 to 20 cc. of 1 per cent solution of procaine hydrochloride. The swelling may not clear for several days but the pain may be quickly relieved. This procedure will sometimes require repetition.

The affected testicle should be supported in a comfortable position and the patient usually requires complete bed rest and the use of aspirin, codeine, DEMEROL,® etc., for relief of pain.

Meningo-encephalitis—The signs of central nervous system involvement require complete bed rest. A single spinal puncture with the relief of pressure will frequently induce prompt and continuing relief of symptoms. The patient may require aspirin, codeine, demerol, or phenobarbital for relief of pain. No specific agent has any demonstrable value. Vomiting may be sufficiently persistent to necessitate the treatment of dehydration by intravenous fluids, dextrose and electrolytes but it is well to avoid undue enthusiasm in rehydrating these patients. As in any other intracranial disease characterized by increased pressure, the rapid introduction of large amounts of fluid may cause sudden and severe pulmonary edema. Relief from pressure by spinal puncture and symptomatic measures for discomfort will usually be followed by steady relief of symptoms and complete recovery.

Oophoritis—The symptoms and sequelae of ovarian involvement are less prominent than those caused by testicular involvement, perhaps because the ovary is not invested with tightly limiting membranes. Treatment should be directed to the relief of pain and discomfort.

Pancreatitis—The abdominal pain and vomiting occasioned by pancreatitis may be sufficiently severe to necessitate sedation and the administration of intravenous dextrose and saline solutions. It is usually unnecessary to continue these methods for more than 24 to 48 hours before recovery begins. Intravenous fluids should be given slowly and in moderate amounts.

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Treatment of Bronchiectasis

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WALDBOTT

IN treating a disease, our first efforts must always be directed toward elimination of its cause. The cause of bronchiectasis and the mechanism of its development are not fully known. Pulmonary infections leading to weakening of alveolar walls, obstruction of the terminal bronchi by mucus and the sudden explosive pressure from coughing are believed to be principally responsible.

The onset of bronchiectasis in children is frequently associated with protracted unresolved pneumonia, pertussis, foreign bodies in the bronchi, and chronic sinus disease. Figure 1 illustrates a condition which probably constitutes one of the chief conditions inducing bronchiectasis, namely, a pneumonitis resulting from protracted obstruction of a bronchus by mucus. Contrary to general belief, allergic asthma does not cause bronchiectasis (Waldbott[1]). It is true that, at the height of asthmatic attacks and particularly during the asthmatic state, there is a temporary widening of fine bronchi in one or more pulmonary segments (Figure 2). This resembles bronchiectasis but is reversible and clears up promptly with allergic management.* The problem is further complicated by the fact that children with bronchiectasis manifest wheezing, cough and expectoration, which simulate asthma. Bronchiectasis is therefore easily confused with allergic asthma.

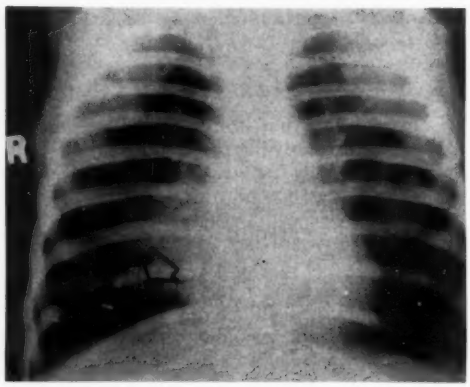

FIGURE 1. Prebronchiectatic state. This pneumonitis of six months' duration was due to obstruction of a bronchus with mucus. Bronchoscopic aspiration and dilation of the bronchus induced a prompt cure.

SEGMENTAL LOBECTOMY

In bronchiectasis, portions of the lungs are destroyed. Furthermore, the diseased parts of the lungs harbor infectious material and serve as a source of infection for healthy lung tissue. Therefore, surgical removal of the diseased portion of the lung constitutes the treatment of choice.

The best surgical practice is segmental resection, i.e., removal of only the diseased segments of one or more involved lobes. By the older method of resecting a total lobe or a whole lung, much healthy lung tissue might be unnecessarily sacrificed. Regardless of the type of operation, enough lung tissue must be left so as not to impair the function of breathing—at least two remaining lobes must be in a healthy condition. Furthermore, there must be reasonable assurance that the original source of the condition has been eliminated so that the remaining lung tissue is not endangered. This implies that in some instances it is necessary either to permit some diseased tissue to remain or to resort to nonsurgical measures only.

The prerequisite for successful surgery is the clear demonstration of bronchial dilations and of their exact extent by well made bronchograms. Their persistence can only be determined if bronchographic studies are carried out on two or more occasions, at least six to eight weeks apart. In Figure 2, a "bronchiectasis" located in one segment of the left lung had disappeared completely eight weeks later while another segment of the right lung showed a similar lesion.

The following preoperative practices are recommended: For two to three days, the child should be given antibiotic treatment and postural drainage. If the latter does not produce much expectoration, a bronchoscopic aspiration is indicated. Particu-

*We suggested the term pseudobronchiectasis for this condition differentiating it from irreversible bronchiectasis.

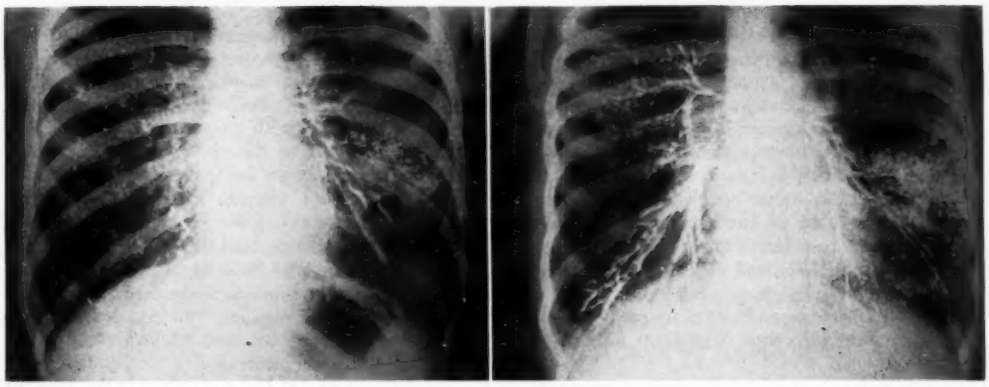

FIGURE 2. "Pseudobronchiectasis," a common feature in bronchial asthma. *Left:* Note the bronchial dilation in left lower lobe on bronchogram. *Right:* Eight weeks later there was complete spontaneous clearance of this area, but a similar process in the right lower lobe. Surgery would have sacrificed these segments unnecessarily.

Illustrations from Waldbott, G. L.: Bronchoscopic therapy in allergic asthma. J. Allergy 20:335-343 (September) 1949.

larly, the lungs should be cleaned of any iodized oil remaining from a previous bronchogram. Narcotic drugs which eliminate the cough reflex should be avoided.

PALLIATIVE TREATMENT

If the process is too extensive for surgical intervention, palliative treatment is indicated. It is aimed at keeping the bronchi (1) free from obstructing mucus, (2) free from infection and (3) free from irritating sources entering the lungs with the inhaled air.

Elimination of mucus from bronchi: *Bronchoscopic lavage*—The thick purulent mucus present in the ectatic bronchi causes the distressing cough, wheezing and dyspnea in children with bronchiectasis. The most effective way of removing it is bronchoscopic lavage. In young children, a general anesthetic is needed for this procedure; in older children, administration of a barbiturate might suffice. The bronchoscope is inserted into the bronchi, the mucus is aspirated and either saline solution or an antibiotic such as penicillin (100,000 units per 20 cc. of saline) is employed to flush out the bronchial tree. In this manner, all but the smallest ramifications of the bronchi can be cleared of mucus and spontaneous evacuation of the remaining ones is induced. The benefit derived from this procedure may last for weeks, even for several months.

Unpleasant accidents from this procedure can be prevented if narcotics are strictly avoided and if we guard against sensitivity to local anesthetics and other drugs.*

*For further details see Waldbott, G. L.: Bronchoscopic therapy in allergic asthma. J. Allergy 20:335-343 (September) 1949.

Postural drainage—A somewhat less effective means of providing elimination of mucus from the bronchi is postural drainage. Nevertheless it is by far the most important routine procedure, since it is much less disturbing to a child. The patient assumes a position of hanging from the hips over the side of the bed or table, the chest being in a near-vertical position. He supports himself by his elbows on the floor or can use a footstool or chair as a rest for his head. After he has been in this position for 5 to 10 minutes, the mucus begins to loosen up, initiating cough and some dyspnea. At this point, deep breathing and easy coughing should be encouraged. It may take from 30 to 60 minutes before the bronchi are adequately evacuated. Preliminary administration of a bronchodilating drug such as aminophylline, epinephrine or an expectorant such as ammonium chloride or syrup of ipecac enhances the elimination of mucus.

Bronchial catheter—Some clinicians have recommended the insertion of a catheter into the trachea for the purpose of aspirating the mucus. This may be useful if the child is cooperative.

Control of infection—Antibiotic therapy is another most essential part in the treatment of bronchiectasis. We must avail ourselves of all known antibiotics. Their choice depends on the determination of the prevailing organism by sputum cultures. While there is a great deal of supporting literature about inhalation of penicillin by aerosol, many clinicians prefer intramuscular injections to inhalation therapy.

In order to determine whether or not sensitivity exists, a preliminary intradermal skin test with a small fraction of the dose should always be made before starting a course of penicillin. It should be borne in mind that in planning a second course,

a time interval of between two and six weeks should be avoided since this favors the development of sensitivity to penicillin. Dihydrostreptomycin by nebulization or preferably by injections, CHLOROMYCETIN,® aureomycin and terramycin are most effective in the control of bronchial infection; sulfonamide drugs have a definite place in the treatment of bronchiectasis.

Occasionally, the mucus of bronchiectasis discloses Vincent's bacillus and spirochetes and, on rare occasions, pure cultures of molds, especially of *Monilia* and yeast. For the former, injections of neoarsphenamine may be given; certain fungous infections respond to large doses of iodine by mouth or to intravenous injections of sodium iodide (10 per cent solution).

The time-honored method of attempting to immunize the system by administration of autogenous bacterial vaccines has been abandoned by some in view of the more modern treatment with antibiotics. However, I feel that vaccines are indicated in bronchiectasis provided the doses are sufficiently diluted so as not to cause unpleasant reactions. Our experience is as yet too meager to determine the efficacy of ACTH and cortisone in relieving bronchiectasis.

"Pulmonary hygiene"—Patients with any pulmonary disease are susceptible to irritation from dust-laden air, from strong irritating odors such as perfumes, cigaret smoke and from sudden change of temperature. Pillows or mattresses should therefore be sealed with plastic covers, rugs and carpets removed from the child's room, the heating outlets covered with damp cloths and the home kept free from dust. Homes with damp basements favoring the growth of fungi, and regions near industrial plants, barns, stables, bakeries, packing houses, etc., should be avoided. Installation of air conditioning in the home may be considered. The child should guard against sudden exposure to cold air. Whenever possible, removal to a dry warm climate is recommended.

The demonstration of a nonoperable bronchiectasis does not rule out the chances of a permanent cure in a child afflicted with this disease. Pathologists frequently encounter bronchiectasis as an incidental finding at autopsy in patients who had had no symptoms, the bronchi being entirely free from mucus. There is reason, therefore, to assume that these children may become well if infection is controlled, the mucus evacuated and the diseased portions of the lungs removed.

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Hemorrhagic Disease of the Newborn

A. H. PARMELEE

BEVERLY HILLS, CALIFORNIA

A LATENT bleeding tendency is a physiologic peculiarity of every newly born infant during the first five to six days of its life. From the first to the third day there is normally a mild prolongation of coagulation time and of bleeding time; after the third day coagulation and bleeding times gradually decrease and reach normal values by the sixth day, as a rule.

This physiologic tendency may under certain conditions assume pathologic proportions and become the underlying cause of more or less serious hemorrhages. This we speak of as hemorrhagic disease of the newborn.

The exact etiology of the physiologic latent bleeding tendency is not known. Neither is it known what precipitates actual spontaneous hemorrhage in cases of true hemorrhagic disease. The discovery that there is regularly a marked fall in available plasma prothrombin during the first three days after birth and that this returns spontaneously to near normal values by the sixth day, made it seem probable that hypoprothrombinemia was the chief etiologic factor. There are, however, disturbing inconsistencies between clinical manifestations and laboratory findings. While hemorrhagic disease does occur in the presence of hypoprothrombinemia, this is not always the case; extremely low plasma prothrombin levels are not always accompanied by hemorrhage, and hemorrhagic disease is not always cured by raising the prothrombin level to normal through administration of vitamin K, nor prevented by maintaining prothrombin at a normal level through prophylactic administration of vitamin K to the mother during labor or to the infant after birth. The indications are that another factor or other factors as yet undiscovered are operative in hemorrhagic diseases.

In our present state of uncertainty it would

seem best to consider hemorrhagic disease of the newborn as due to a combination of factors, some having to do directly with blood coagulation, and some with blood vessel wall integrity.

Many hemorrhagic manifestations occur in the newborn, most of them inconsequential. There does not seem to be uniformity of opinion about what hemorrhages are and what are not manifestations of hemorrhagic disease. The definition proposed by Clifford[1] is in my opinion the most satisfactory in light of our present knowledge. It is: "the syndrome of spontaneous external or internal hemorrhage accompanied by hypoprothrombinemia occurring in the newborn, excluding that associated with trauma, asphyxia, or some definite disease."

EVALUATION OF TREATMENT

The treatment of hemorrhagic disease is difficult to evaluate, first because clinicians do not use uniform criteria regarding what is and what is not hemorrhagic disease, and second, because in the majority of cases bleeding tends to stop spontaneously and whatever treatment has been used sometimes receives unwarranted credit. Reports from various sources have, therefore, been conflicting. The conflict arises largely from the uncertainty of etiology. Those satisfied to accept hypoprothrombinemia as an essential and determining causative factor have a strong case in favor of the use of vitamin K both as prophylactic and as curative treatment.

It is well established that hypoprothrombinemia of the newborn can be prevented either by giving vitamin K to the mother during labor or to the infant at birth, and that an existing hypoprothrombinemia can be promptly corrected by giving vitamin K. In addition there are some clinical reports that indicate a lower incidence of hemorrhagic manifestations where vitamin K was routinely used as a prophylactic measure.[1-3] On the other hand those who are not convinced of the essential importance of this factor have considerable evidence that casts doubt on the efficacy of vitamin K in preventing or curing hemorrhagic manifestations. There have been several reports from large centers to the effect that hemorrhagic manifestations occurred with about equal frequency whether the prothrombin level of the blood was low or high, and whether the infants or their mothers were given vitamin K or not.[4, 5]

Contradictory reports on the value of vitamin K as prophylactic or curative treatment is confusing to the clinician. But from a practical standpoint he need not be too much concerned since there is complete agreement on the harmlessness of vitamin K and its cost is low. The consensus is that to be on the safe side it should be used. Many obstetricians give it routinely to the mother during labor, and, in cases where delivery has been difficult and in premature deliveries, to the infant at birth as well.

The wisdom of relying on vitamin K alone in the curative treatment of hemorrhagic disease is questionable. Certainly such a course would not be safe if the bleeding is of a serious nature. In such cases there is strong possibility that in spite of successfully raising the prothrombin level to normal the bleeding will persist; I have twice had that experience. In that case valuable and perhaps vital time will have been lost. In all cases of severe or even moderately severe hemorrhage a transfusion should be given immediately whether the bleeding is known to be due to hemorrhagic disease or not.

PREVENTION OF HYPOPROTHROMBINEMIA

The prevention of hypoprothrombinemia is accomplished either by giving the mother one of the vitamin K-like synthetic preparations, 1 mg. daily by mouth during the last two weeks preceding delivery or by giving her 4 mg. intramuscularly during labor, preferably two to four hours before delivery. In case the mother has not been given vitamin K in this manner, prevention can still be accomplished by giving the infant 1 to 2 mg. intramuscularly immediately after birth. If the infant is premature or the delivery is a difficult one, many advocate giving vitamin K to the infant even though the mother has been treated.

The dosage of vitamin K in the active treatment of hemorrhagic disease is 1 to 2 mg. intravenously or intramuscularly. When given intravenously, prothrombin levels will be adequately raised in one to two hours. The action when given intramuscularly will be slightly slower.

TRANSFUSIONS

When transfusions are indicated, fresh whole blood should be given intravenously in the amount of 10 to 15 cc. per pound body weight; the usual amount given is 50 to 100 cc. Proper typing is necessary, and for female infants it is important that the blood be of the same type and Rh factor. In some instances the transfusion will need to be repeated in twenty-four hours. It may even be necessary in rare instances to repeat the transfusions daily for several days. Repeated transfusions are usually indicated when the blood loss has been excessive.

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Scientific Exhibit

Technic of Tracheotomy

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TRACHEOTOMY is one of the oldest surgical procedures, dating back to the second century, B.C. The primary purpose was the relief of respiratory distress due to laryngeal obstruction. It is now being employed more and more to provide an easy means of keeping the tracheobronchial tree free of normal and abnormal secretions. Tracheotomy has been useful in infants with an overwhelming infection of the tracheobronchial tree. The same use is employed in infants or small children in whom such an infection is the result of a vegetal foreign body.

A new use is in cases with a loss of or a diminished pharyngeal reflex so that salivary secretions enter the trachea instead of the esophagus as in bulbar poliomyelitis, tetanus and dermatomyositis.

Infants with bilateral choanal atresia are helped through a difficult period of breathing and swallowing by the use of a tracheotomy. The surgical relief of the nasal obstruction is made easier and less dangerous because of this added airway.

SIGNS AND SYMPTOMS OF LARYNGEAL OBSTRUCTION

1. Retraction of the supraclavicular spaces, suprasternal notch, intercostal spaces, and epigastrium.
2. Increase in the respiratory rate.
3. Increase in the pulse rate.
4. Elevation of the temperature.
5. Restlessness.
6. Pallor.
7. Cyanosis.

Adapted from an exhibit shown at the American Medical Association, San Francisco meeting, June, 1950.

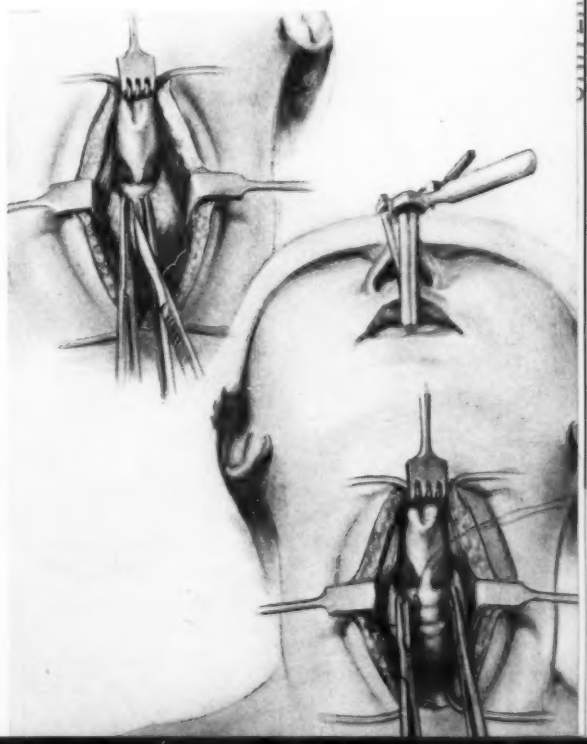
Introduction of a bronchoscope *(right)*, particularly in infants, provides a rapid airway so that tracheotomy can be performed in an orderly manner. The relief of the breathing difficulty helps to prevent the introduction of air into the mediastinum from around the trachea. The bronchoscope serves as an excellent guide to the trachea in a short, fat neck. The one disadvantage is that the decannulation of the trachea may be delayed a day or two because of the additional trauma to the larynx.

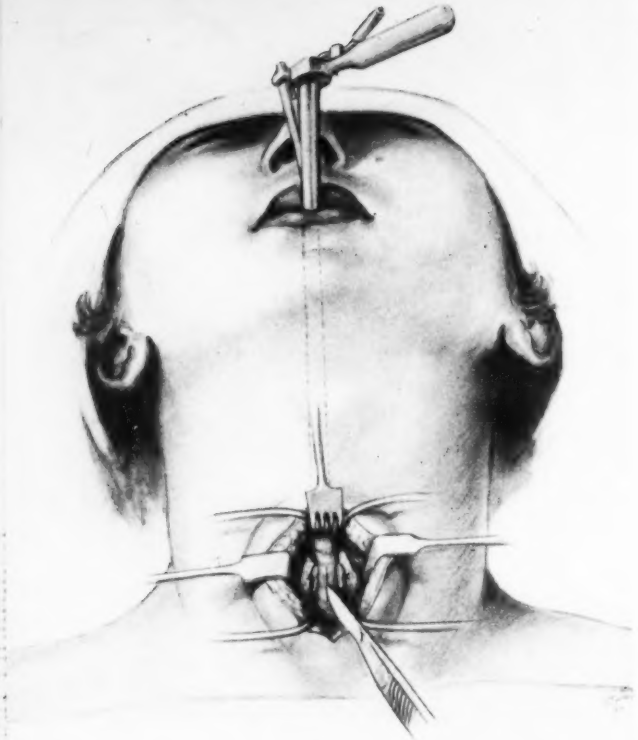
The collar incision usually results in a less noticeable scar and prevents the pulling sensation sometimes produced by a midline scar. The midline incision is the one of choice in an emergency procedure because of the absence of important blood vessels. The collar incision offers a distinct advantage in performing laryngofissures and laryngectomies.

(Below) The isthmus of the thyroid gland is divided in the midline if it cannot be retracted out of the way.

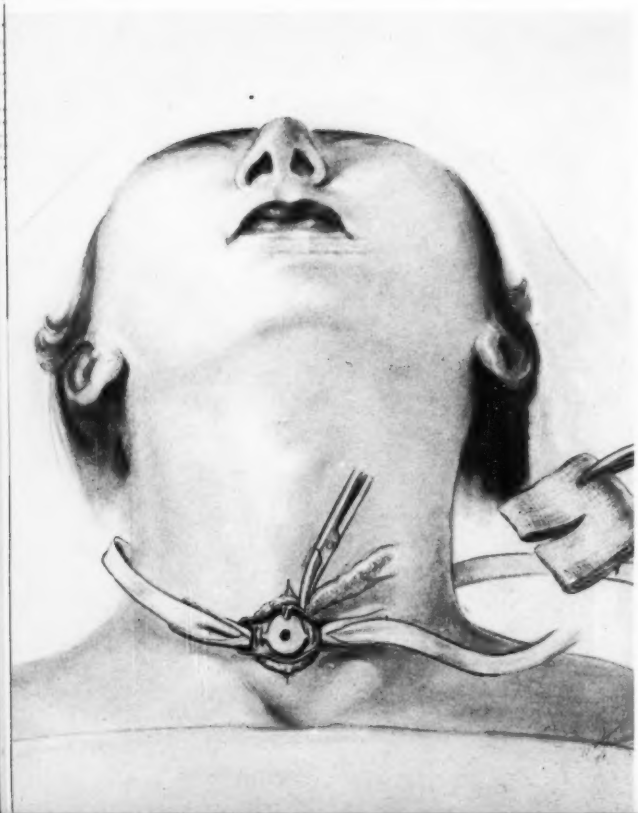
The method of clamping, dividing and ligating the divided ends of the isthmus is shown.

(Below) The incision has been extended to show the thyroid cartilage, the cricoid, and the trachea. The division of the isthmus and the ligation of the cut ends is repeated. It is important to refrain from skeletonizing the trachea or opening the fascial plane more than necessary so as to prevent the entrance of air into the mediastinum with subsequent pneumothorax in some cases (Forbes).

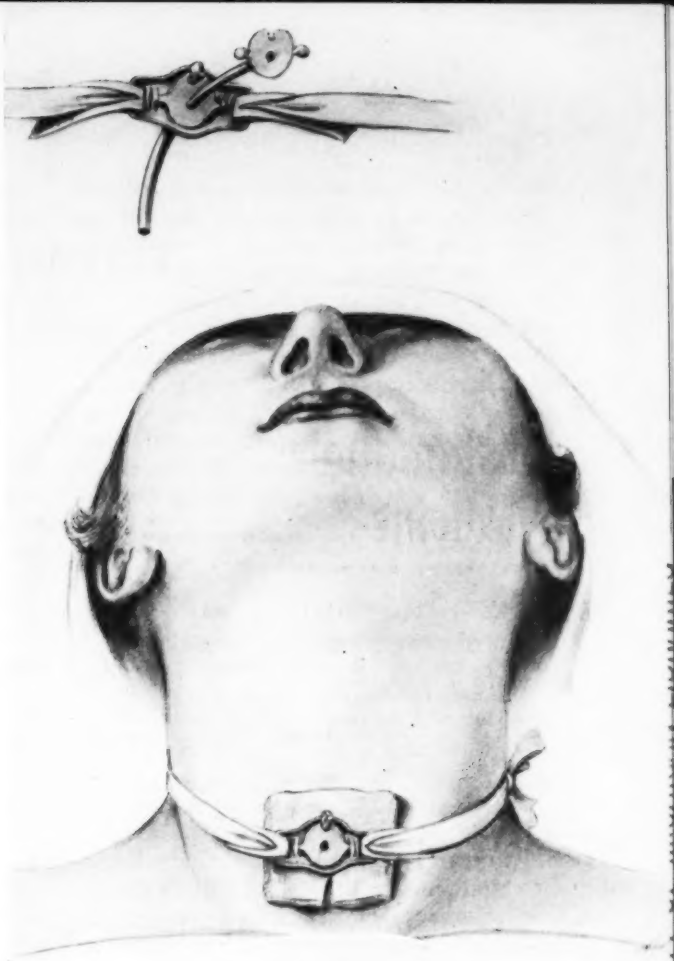




The use of the bronchoscope to provide a source of air while performing a tracheotomy is of great advantage. The bronchoscope serves as a guide to the trachea in short, fat necks. Relief of the breathing difficulty helps prevent the sucking of air into the mediastinum from around the trachea. After the procedure has been completed, this complication is avoided further by the packing of the wound with a gauze strip dusted with one of the sulfonamides.



The snug packing of a gauze strip dusted with one of the sulfonamides (Tucker) prevents secondary infection of the operative wound and helps to seal off the mediastinum, preventing the complication which was described previously.



The first tracheal ring should not be divided because of the danger of injury to the cricoid cartilage and resultant stenosis of the larynx. The cricoid cartilage is the only complete cartilage in the larynx and perichondritis may cause a reduction in the size of the lumen.

The tracheal rings must be divided with care if a bronchoscope is not in place because injury to the posterior wall of the trachea by a plunging knife may cause serious complications.

POSTOPERATIVE CARE OF TRACHEOTOMIZED PATIENTS

The primary consideration is to keep the "airway open." This is accomplished by the frequent aspiration of secretions from the tracheobronchial tree. The inner tube provides an easy means of keeping the tracheotomy tube open. Antibiotics are used as indicated. Moisture content of the air must be increased by the use of cold or hot steam. Wetting agents (Gorman) such as SANTOMERSE* increase the wetness and prevent crust formations. This can be added to the source of steam and can be introduced directly into the tracheobronchial tree. If crust formations occur, bronchoscopy may be necessary to remove them.

*Monsanto Chemical Company.

EDITORIALS

Postgraduate Medicine FOR THE CONTINUING EDUCATION OF THE PRACTICING PHYSICIAN

Editor-in-chief: CHARLES W. MAYO, M.D.
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APRIL 1951

THE SCIENTIFIC METHOD IN MEDICAL RESEARCH

Many historians place the beginning of scientific research in medicine as coincidental with the publication of William Harvey's "De Motu Cordis" in 1628. He urged repeatedly the experimental method. Following William Harvey the methods were reintroduced only by such classical studies and observations as those of John Hunter and Edward Jenner. Here was the beginning of immunology. Another landmark was the work of William Beaumont with his systematic experimental studies of the functions of the gastrointestinal tract. Sir Henry Dale in his notable address on the "Scientific Method in Medical Research," published recently in the *British Medical Journal*[1] calls attention further to the basic contributions of Claude Bernard, to the work of Karl Ludwig in Leipzig, and to studies of Louis Pasteur, Robert Koch and Joseph Lister.

The rise of the laboratory brought with it a decline in the importance of research in the wards and rooms of the hospitals. Sir Henry Dale characterizes it as a "growing tendency to aloofness of interests and aims and a progressively dominant assumption that experimental research, and indeed the attempt to apply scientific criteria of evidence with any rigidity, was a matter for the laboratories." Yet at the same time many leaders in medicine were depreciating the contributions of the laboratory; John B. Murphy is credited with having sneered at diagnoses made "by glassware instead of by brains." Sir Henry Dale remarks that there was a tradition in his student days that a physician should use no apparatus that could not be carried in his pocket or in his conveniently rigid and capacious tall hat. Even the thermometer was suspect as interfering with the doctor's ability to determine the presence of fever by feeling the patient's skin with his hand.

Perhaps the delay of the clinicians in adopting the scientific method in medical research may be a result of the traditional respect for medical authority which came down from Hippocrates through the 1500 years thereafter in which medicine languished while men worshiped the writings of Galen and Hippocrates until the great Paracelsus tossed them into the flames. In 1840 Sir Thomas Smith recommended cod liver oil for the rickety lions in the zoo in London but it remained for the medical profession to wait until 1910 to begin to abolish the condition in human beings with the same substance.

Sir Henry Dale rightly credits Sir Thomas Lewis' leadership for developing a proper status for clinical research. The greatest influence, however, has come from the devel-

opment of biochemistry which is essentially a product of the present century. From chemistry came the basic research of Emil Fischer, Frederick Gowland Hopkins, Paul Ehrlich and, more recently, such triumphs as that of Kendall and Hench. We are just beginning to realize the profound importance of biophysics, the sister science of biochemistry, as a technic for the solving of many human diseases.

Fundamental to the scientific method is the science of statistics. "The calm and ruthless logic of the statistical method," says Sir Henry Dale, "has had an influence of inestimable value in giving a higher level of scientific authority to the planning of investigations and to the appraisement of their results over the whole field of medical research."

Great Britain has been confronted by the same problems of administration and control that have concerned us in the United States in the allocation of governmental funds for medical research. Sir Henry Dale, who has for many years been a director of the Medical Research Council in Great Britain, speaks from his wealth of experience in behalf of scientific control over grants of money. "Our chief concern," he says, "must be to ensure that the public funds directly granted for medical research continue to be administered and allotted, as hitherto, by a Council composed predominantly of experts who know the meaning of research, and who can be trusted to recognize the ways in which its truly scientific pursuit can most effectively be encouraged, without either facile enthusiasm or obstinate caution."

Sir Henry is well aware of the great dangers of political pressure. "Success in medical research," he points out, "very often depends on an investigator's freedom to seize and to follow an unexpected clue, even when it appears to lead far from the original objective, and in a direction offering no visible likelihood of practical developments." He concludes: "While the administration remains safely under such expert and independent control, the support of medical research from the public funds, now an indispensable condition of its pursuit in this country on the present progressive scale, will continue to be protected from any serious danger to its freedom and integrity. Such danger could arise only if the distribution of the available funds were to become subject to political pressure, seeking to influence its direction in order to satisfy some popular clamour or sentiment, to meet some official convenience, or to enhance some Minister's prestige."

MORRIS FISHBEIN

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FOUNDATIONS, CORSETS AND BRASSIERES

In an editorial in *Postgraduate Medicine* for February[1] I expressed my annoyance with a questionnaire circulated by Fact Finders Associates, Inc., dealing with the supporting garments worn by women. The questionnaire, readers will remember, requested intimate information as to the concealed garments worn by my employees. I ventured to reproach the agency mildly for using a dubious and circuitous route in an effort to establish the essentiality of foundation garments, corsets and brassieres for health. This soft impeachment aroused a veritable furor of excitement which caused me to undertake a research into some of the scientific documents that have been developed from time to time by the medical profession in their desire to be helpful in the evaluation of support to body tissues as a means of avoiding unnecessary aches and pains.

In 1910 an eminent gynecologist and an orthopedic surgeon combined to present their report[2] of an experimental study of certain phases of chronic backache before the American Gynecological Association in New York, the American Orthopedic Association in Hartford, the Gesellschaft der ehemaligen Hoffa'schen Assistenten in Berlin, and the Anglo-American Medical Association in Berlin. The problem that dis-

turbed them was naturally low back pain, a condition which has been called from time to time weak back, uterine backache, irritable spine, chronic lumbago, railroad spine (when it affected traveling men) and, scientifically, relaxation of the sacroiliac joints. The scientists suggested as an alternate name "static backache."

In their desire to approach the subject from the important viewpoint of research, a technic was devised for determining the center of gravity of the human body in the erect position and to detect the changes that developed with the human being in other postures. In the research "female nude professional artists' models" from the school of the Boston Museum of Fine Arts cooperated. Many of them were observed for three and a half years.

The research served to establish the fact that the center of gravity of the body with the human being in an erect position lies in front of the ankle joints, in front of the knees and anterior to the sacroiliac joints. If for any reason the center of gravity becomes displaced forward, the strain on the muscles of the back becomes increased. Mere maintenance of the standing position results, after a short but variable time, in slumping of the body to what has been called "a fatigue position."

The investigators found that most corsets worn in 1910 had little if any appreciable effect on body balance. The worst corsets were those which were long behind and short in front, exerting their greatest pressure at the waist and with a severe curve in the back. Good corsets were short behind especially at the top and relatively long in front especially at the bottom. Good corsets invariably moved the center of gravity back. Under their influence the shoulders were thrown back of the normal but the hips never moved further backward than did the shoulders.

From their observations Drs. Reynolds and Lovett agreed that proper corsets helped to relieve low back pain by serving as a splint and a support; they tend to correct vicious balance by carrying the center of gravity backward, thus relieving muscular strain; they partially splint the lower back and they serve as an artificial ligament to the glutei muscles. Indeed they found that many cases of backache were relieved by the use of corsets and high-heeled shoes alone. They felt that any attempts to define this condition as a disease were unfortunate because the condition is a mere symptom complex "due to an abnormal attitude induced by the peculiarities of the skeleton, lack of proper muscular balance or abnormal conditions in the abdomen or elsewhere." Obviously such conditions as displaced disks, degenerations or fractures of vertebrae are in a different category.

In 1941 Dr. Frank Ober[3] reviewed the article by Drs. Reynolds and Lovett and affirmed that the fundamental principles which they established were just "as true today as they were 30 years ago." A corset which does not fit the patient properly will not help a lame back but may actually increase backache. The function of a good corset is support of weakened or relaxed tissues and restoration of balance.

Most of the trouble with human mechanics arises from the fact that the human being was evolved from an animal walking on four legs to one that walks erect and on two legs. The spinal column of four-legged animals has a concavity in the back. When the human being stands up the spinal column develops curves and new tensions. The change to the erect posture has brought about difficulties with the feet, the knees, the hips and the back.

Unfortunately the makers of various supporting devices for the portions of the body structure, including the body wall, where the greatest stresses occur, have not undertaken sufficient scientific research to establish the form which supports should take. Certain occupations throw extra stresses on portions of the body and are more likely than are other occupations to result in revolt which the body expresses by aches and pains. Exercises are helpful in strengthening weak-

ened tissues or areas where stresses are beyond the capacity of the tissues to support but until the tissues have developed sufficient strength, extra artificial support may be necessary. We help weak eyes with glasses, insufficient hearing ability with hearing devices and weakened legs with crutches. Foundation garments and corsets are helpful in providing artificial support for the back and the abdominal wall in carrying the great load which they support when the human being stands erect.

Body mechanics is a specialty of medicine which deserves far more scientific study and research than it has yet had. Most of our approach to the subject has been empirical and all that the survey of Fact Finders Associates accomplished was to emphasize these empirical opinions. Last month they circulated the results of their questionnaire which indicated that 84 per cent of doctors, nurses, personnel directors of retail stores and industrial plants felt that properly constructed and fitted corsets, girdles and brassieres contribute greatly to the good health of American men and women. No doubt it is a fact that 55,000,000 American women above 15 years of age wear foundation garments and no doubt they wear them because they like them and because they are convinced they make them feel better. No one will question that properly fitted foundation garments, corsets and brassieres make women look better, but further scientific studies of the problem are needed to establish conclusively their value in body mechanics.

MORRIS FISHBEIN

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To the Editor:

In the February, 1951, issue of *Postgraduate Medicine* there is an editorial by Dr. Morris Fishbein on Hadacol. In this article Dr. Fishbein states a sum of money reputedly being spent through the advertising agency known as Erwin, Wasey & Company, Inc.

Erwin, Wasey & Company, Inc., resigned the Hadacol account last November. Will you, therefore, kindly print a correction of the statement made by Dr. Fishbein referring to Erwin, Wasey & Company, Inc., in your next issue.

Cordially yours, Walter W. Holt
Erwin, Wasey & Company, Inc.

ACADEMIC HUMOR

In a recent guest editorial on Academic Humor by Dr. Richard B. Dillehunt, Dean Emeritus, University of Oregon Medical School, published by the Oregon *Daily Journal,* Doctor Dillehunt philosophizes in a kindly manner about university presidents and teachers who are so immersed in the academic atmosphere in which they constantly reside that they fail completely in adjustment to the surroundings of which they are at the same time a part. His experience as dean of a medical school has led Doctor Dillehunt to suggest to college presidents that they examine all prospects for positions on the faculty with great care as to the presence or absence of a sense of humor. He feels that the faculties of medical schools, while less tainted by such abstractions as liberalism and rights as are the occupants of nonprofessional campuses, have nevertheless the same prima donna qualities that are typical of practically all teachers.

The Emeritus Dean is not an advocate of ebullient excess humor or the performance of practical jokes or the perpetuating of low-grade whimsies, but he does feel that "the lack of a sense of humor with the usual coeval poor sportsmanship is more reprehensible in a college organism than in some evanescent socio-political brainwave so common to the intelligentsia therein."

I gather from this that my friend and classmate of the first decade of the present century is becoming a little bored by the innumerable societies, organizations and associations and similar groups devoted to reforming, revolutionizing and changing much that has been accepted as established procedure in living. Some 50,000 years of intellect have been enjoyed by mankind since, according to the anthropologists, the human brain first reached its present state of development. Since only a small percentage of mankind has reached the stage of being able to laugh at themselves and to realize their own absurdities, another 50,000 years may be needed to bring the mass up to that level of tolerance.

MORRIS FISHBEIN

Men of Medicine

"The attrition of young minds upon . . . orthopedic surgery"

THESE days, fewer children in braces are seen hobbling along the streets or hanging back in wide-eyed hurt from their agile playmates in school yards. Fewer men and women have to buckle on appliances before starting their day's work. During the past half-century the "brace and buckle" era of orthopedics has, for the most part, disappeared, to be replaced by more daring surgery. In that gradual change, Dr. Arthur Krida—fisherman, forester and topflight surgeon—has had a formative part; his hands, strong enough to manipulate bones or land a record-breaking salmon, have helped shape the new pattern. He is careful of his blunt, sensitive hands; he is annoyed by an ink smudge on his fingers, and immediately washes them; disturbed by rough finger nails and quickly files them smooth.

Dr. Krida himself admits modestly that he has "pushed an oar here and there" to bring about that change in the field of orthopedics during his 40 years of medical practice, 30 of them in orthopedic surgery. He has been able to watch and to learn from the earlier masters, and to carry on as a pioneer himself in operations that have enabled many hundreds—he has lost count—to walk or to use their arms and hands again.

Brace shops at one time just about dominated orthopedic hospitals, Dr. Krida recalls (the "i" in Krida is pronounced like the "e" in orthopedic) and now, though still important, they are more nearly accessories. The aim of present orthopedic surgery, which leaped ahead of the limping crutch-and-brace school, is cure, not palliation. Whether it is a case of congenital deformity in a child or of a crippling industrial accident in an adult, the surgeons try to rid the patient of his brace. Earlier, deformities from the big toe to the neck were treated by mechanical appliances because orthopedic surgeons of bygone days, with few exceptions, had limited surgical experience, or skill. Surgeons have now discovered the limitations of bracing and of splinting and the use of appliances in general, and have learned how much more quickly surgical intervention can get a patient back on his feet.

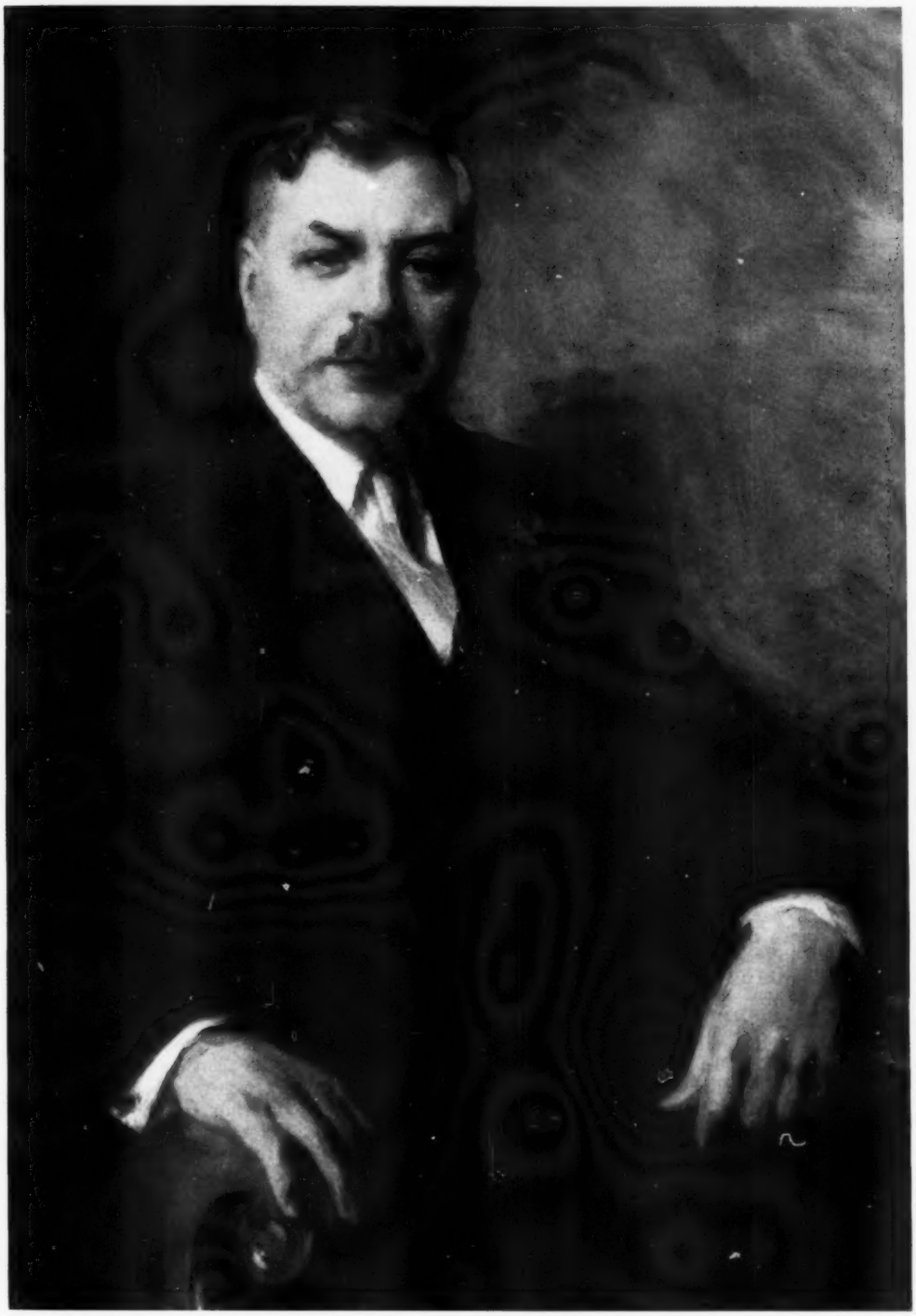
Dr. Krida has also seen, during his career, the disappearance of the term "railway spine" for a complaint common to those who have been in railroad accidents. The condition is now called "litigation spine" or, more popularly, a neurosis. In the army the victim is sometimes known as a "gold-bricker."

Dr. Krida explains all this with a gruff voice that can be as blunt with stumbling students as it is gentle with crippled patients. His occasional bone-breaking rebukes of erring students became a Bellevue Hospital byword. Doctors now practicing on their own can recall how he sand-blasted one student in an orthopedic quiz section. His shattering reply to an attempted bluff was, "Doctor, that's a lot of sheer moonshine. You'll find that a little orthopedic reading is worth reams of your speculative ratiocination."

But when he handed in his resignation, effective last fall, as professor of orthopedic surgery at New York University and Bellevue Medical College and as director of orthopedic surgery at Bellevue Hospital, his former interns, residents and associates got together to give him a beautifully bound testimonial book containing more than two-score handwritten tributes praising him for his faithful attendance at rounds, lectures, conferences and operations, and for the way "the Chief" was "patient and paternal" and trusted his associates to run their services in their own way—while standing by, ready with counsel when it was needed. And he has had the same secretary for 27½ years.

Dr. Krida held the hospital and medical school positions for 20 years. The first professor of orthopedic surgery at Bellevue, Dr. Lewis A. Sayre, held the post for 40 years (1860-1900), his successor and son, Dr. Reginald H. Sayre, whom Dr. Krida succeeded, for 30 years (1900-1930). Dr. Krida, husky and happy in retirement from teaching but by no means from practice, remarks: "The younger generation can't take it."

Unlike many doctors, Dr. Krida cannot point to any strong personal experience that drew him toward his specialty. He was born January 6, 1888, in Lodz, in the western part of Poland, the son of a family with a cosmopolitan background. His father, a citizen of Austria, was a Czech; his mother was German, and German was the language of the home. He was brought to this country at the age of five; he has little memory of his



He has "pushed an oar here and there . . ."

(From a painting by Irving S. Wiles)

early childhood in Poland and never has had a desire to return for a visit. The family—Dr. Krida describes his father as an "artisan"—settled in upstate New York. He had little early formal schooling and got most of his education at home.

His first interest in medicine came in "Horatio Alger, M.D." fashion from working as an office boy for a physician in Schenectady, New York. Occasionally, when the doctor made his rounds, he let young Krida drive the buggy for him, and the boy listened eagerly to the older man's descriptions of disease. He decided that a doctor's life was a good one and an interesting one. He spent much of his idle time in the doctor's office studying medical books. With little more formal help than this, he passed his examination for Albany Medical College, from which he received his M.D. *cum laude* in 1911. He started in private practice in Schenectady and from 1914 to 1916 he was adjunct professor of physiology at Albany Medical College; in 1917 he was named demonstrator of anatomy.

World War I provided the chance that turned him definitely toward orthopedic surgery, a field in which his interest had been growing while at Albany. In 1917 he was commissioned a first lieutenant in the U. S. Army and promptly lent to the British, who were short of doctors. En route to England on the troopship he heard that orthopedic surgeons were in especially great demand; they were needed to patch up the most severely wounded. When he heard of the shortage, he thought: "That's what I want!" and volunteered for that service.

He spent the years 1917-1919 at Oxford, England, working with the wounded, but saw no active service. He found that British orthopedic surgery was "very high grade." He had the chance of working with the famous British surgeon, Dr. Gathorne R. Girdlestone, and later kept in touch with him until the latter's death last December 30. Shortly after hearing of the death of his friend Dr. Krida received a Christmas card which had been addressed before the holidays; its scrawled message said that Dr. Girdlestone was presently at St. Bartholomew's Hospital in London for "reconditioning."

Back in this country in 1919, discharged as a captain, Dr. Krida resumed private practice and in 1922 became assistant orthopedic surgeon at the Hospital for the Ruptured and Crippled (now called the Hospital for Special Surgery) in New York City. There he began a close association with Dr. Royal Whitman, one of the best known orthopedic surgeons in the United States, for whom the famed clinic at the hospital is named. The association lasted until Dr. Whitman's death, at the age of 90, in 1946.

War-time experience alone may make a surgeon's skill one-sided; in Dr. Whitman's service he rounded out his experience with reconstructive war surgery, broadening his knowledge of shell and machine gun wounds by studying the problems encountered in civilian practice. Dr. Krida continued as assistant orthopedic surgeon until 1924, when he became chief of the clinic and associate orthopedic surgeon, serving until 1932. Then, in 1930, he went to Bellevue-New York University.

While at the Hospital for the Ruptured and Crippled and later at Bellevue, Dr. Krida developed the operative technics for which he was to become so widely known. His favorites within the specialty of orthopedic surgery soon became the knee and the hip. His grey-blue eyes light up with an artist's love of his work when he talks about the knee—"I am really very fond of the knee!" He tackled the problem of congenital dislocation of the hip, he says, because not many other surgeons seemed to be much interested, and because it seemed "the most useful" thing he could do; the deformity resulting from a neglected congenital dislocation is one of the most distressing of crippling ailments.

One of his first papers was on the subject of the knee; it was "A General Utility Incision for Exploration of the Knee Joint," published January, 1925, in the *Journal of Bone and Joint Surgery*, and has been widely quoted in medical literature. The next year (May, 1926) he published another paper on the knee, also extensively quoted: "The Operative Treatment of Knee Joint Instability Following Crucial Ligament Injury," in the *International Journal of Medicine and Surgery*. In that paper he revealed his long-lasting sympathy with the crippled and his continuing belief that they should, through surgery, be given a chance to use their limbs normally. He expressed his distaste for the discouragement felt by many surgeons about this knee injury and added: "This writer does not feel that patients with this disability should be *ipso facto* committed to a life of brace-wearing." Dr. Krida has continued to believe that nobody should be needlessly condemned to using a brace or crutch for life.

Dr. Krida looks back on many patients dramatically healed by the operation. There was, for instance, the case of a 21-year-old man who injured his knee aboard ship. He was able to walk only with the help of a cane following treatment with a plaster-of-paris bandage. After operation ("fascioplastic reconstruction of the anterior crucial and internal lateral ligaments") he was able to walk normally—and play football. In another case, a 24-year-old man, who had injured his knee by stepping into a hole in a pier, was unable to walk without the aid of a brace. After the operation he threw away the brace and went back to work.

In the treatment of congenital dislocation of the hip, Dr. Krida has been one of the most impor-

Dr. Arthur Krida (with mask and glasses, instrument in hand) operating at Bellevue Hospital, New York City.

tant advocates of relieving the condition by manipulation rather than by open operation. He reported cheerfully in a paper in the *Journal of the International College of Surgeons* for October, 1940: "The object is the restoration of an anatomically, substantially perfect hip, and such an end-result may be attained in a large majority of cases. . . ." He developed operative technics, not theories. He has not bothered to speculate on why four times as many girls as boys have a congenitally dislocated hip—why, if only one hip is dislocated, it is usually the left—or why certain nationalities and races are more likely than others to be born with the malformation.

Dr. Whitman, in his classic textbook *A Treatise on Orthopedic Surgery,* refers with approval to Dr. Krida's work in improving earlier technics. Never lavish with his praise, Dr. Whitman referred to "a notable advance made by Dr. Krida," and added: "It may be noted that the originality of this procedure is not in any of the details of treatment, all of which are old, but because the correction of the deformity is determined in advance, regardless of the apparent security of the immediate reduction." Dr. Krida owns all nine editions of the work—and believes he is the only individual in New York who can make that boast.

MANY of his operations were, says Dr. Krida, "modifications and integrations" of the work of earlier surgeons. There are, as a matter of fact, few really original operations in surgery, just as there are few really original inventions in the field of mechanics; the men credited with being the originators built on the work of their predecessors. But one operation that "came out of the blue" was his operation on the foot. Boldly he called his paper on this procedure "A New Operation for Metatarsalgia and Splay-Foot," printed in *Surgery, Gynecology and Obstetrics* for July, 1939. He lamented the fact that women prefer narrow shoes,

complaining: "In some occidental communities it seems to be still possible to induce women in the age period mentioned (third and fourth decades) to adopt a wide shoe with a low heel for habitual use. In my community I have found this to be largely unfeasible. . . ." In the history-making operation, Dr. Krida uses fascial bands to repair the damage, and no metatarsal support is needed.

Like many doctors who are called on to correct deformities of the foot, Dr. Krida thinks it would be a fine thing if young children could walk barefoot, or in soft sandals, on uneven ground instead of in hard shoes along smooth floors and sidewalks. If they did, he pointed out in another early paper ("The Symptoms and Treatment of Weak Feet in Children"), published in *American Journal of Surgery,* June, 1926, there would be less work for the orthopedic surgeon when the children grow up. In some cases of weak feet he does advocate use of arch supports—which technically might be considered braces.

Dr. Krida has no pet theories of his own about gout, but remarks with a grin that he knows a man who gets an acute attack of gout every time he has a quarrel with his wife. Gout comes within the field of orthopedic surgery when it becomes necessary to remove the calcareous deposit from a joint. The diagnosis of gout has not been made often in the United States, Dr. Krida says, just because not many doctors are thinking about it. But the recent interest in rheumatoid arthritis has made them more conscious of gout; the disease is no longer in the back of their minds, and they now make the diagnosis more often.

Certain types of arthritis too are in the field of orthopedic surgery. Dr. Krida points out with a bit of acerbity that 15 years ago orthopedic surgeons were about the only medical men who showed much interest in arthritis; their concern was with the prevention of deformity and the repair of damaged joints. It is only recently that medical treatment has come into prominence. Eventually, if ACTH and cortisone work out as well as they seem to be doing in the treatment of rheumatoid arthritis, there may be less work for orthopedic surgeons in the field they once had almost to themselves.

Infantile paralysis victims also may come under the care of orthopedic surgeons. During the past 30 years Dr. Krida has seen a growth of operations for the after-effects of polio, and the steadily decreasing use of braces. About the best that surgery can do in severe cases, says Dr. Krida, is to stabilize the weak member. He explains with poetic imagery: "You can't do much with a definitely paralyzed muscle. The fire is out, and only the ashes are left."

Just what conditions fall into the purview of the orthopedic surgeon depends, of course, on the community, Dr. Krida explains. A general surgeon, for instance, may be called on to do operations of almost any kind if no specialist in the field is available. In New York, "where there is a specialist for the right ear and one for the left," operations for harelip and cleft palate are usually done by an oral surgeon, sometimes by a plastic surgeon.

In his teaching, Dr. Krida has constantly warned students not to forget the patient in their interest in his disease. He considered formal lectures a "necessary task," and as he approached retirement he tended more and more to delegate them to senior associates. He liked best what he calls "the attrition of young minds upon the backgrounds of orthopedic surgery." In his formal lectures to large classes he insisted, with barbed emphasis, on considering the patient as a person: the necessity of getting a careful case history, eliminating irrelevant material, and then making a thorough physical examination. He found that such essentials were frequently "short-circuited," and that students, interns or even residents came to think that medical machinery answered all questions—that putting an x-ray film into a shadow box and making various laboratory tests was enough to give a complete picture of what was wrong with a human being.

Dr. Krida has a favorite anecdote to bring home his point. At one conference of his Bellevue associates the case of a patient with pain in the forearm and wrist was being discussed. An x-ray in the shadow box showed a shrapnel fragment lodged in the lower cervical region. Dr. Krida's associates thought that the obvious way to help the patient was to remove the fragment. Dr. Krida, as usual, insisted on a physical examination of the patient. It proved that he had a stenosing tenosynovitis of the thumb extensor tendon sheath. Dr. Krida ends the story with the ironic comment: "A small operation on the tendon sheath promptly relieved his pain. The metallic fragment in the neck was promptly forgotten."

At present, Dr. Krida does no teaching nor any formal lecturing—"Not if I can help it!"—and he is not writing a book. He is, he explains, "too lazy" to write a book, but he has inspired former students and associates to write books. He is retired only from teaching, not from his private and consulting practice in orthopedic surgery, which he loves. He has developed a kind of amused indignation toward fellow physicians who have heard about his "retirement" and look surprised when they see him, as if to say: "You still around? I thought you'd retired! . . ." He has no intention of going to St. Petersburg to doze on a beach or play quoits in the Florida sun.

He does intend, when he gets around to it, to write some more medical papers. So far he has had more than 30 published, beginning in 1923. The most recent is a chapter on "Acute Suppura-

Fishing in the Restigouche River, New Brunswick, Canada. Dr. Krida is seated in the bow of the canoe.

tive Arthritis" in *Arthritis and Related Conditions,* edited by Dr. Theodore Franklin Bach, published in 1947. All his papers are written in longhand.

He is still on the editorial board of the *American Journal of Surgery.* He describes himself half-seriously as one of the doctors listed on the front page as "front men"; the position does not require much work. He is consulting orthopedic surgeon at Bellevue, the University Hospital, the Hospital for Special Surgery, Knickerbocker, Lutheran and U. S. Marine Hospitals in New York; St. Agnes Hospital at White Plains, New York; Beth Israel Hospital, South Nassau Communities Hospital at Rockville Centre, New York; St. Francis Hospital, Port Jervis, and North Hudson Hospital at Weehawken, New Jersey. He also acts as impartial specialist in orthopedic surgery in workmen's compensation cases.

His interest in arthritis led to his membership in the New York Rheumatism Foundation and for 10 years he has been a member of the advisory committee of the Greater New York Chapter of the National Foundation for Infantile Paralysis. He proudly carries the badge—potent with traffic policemen—of an honorary police surgeon of New York City. A memorial seat in Bellevue's new Alumni Hall has been named after him. He is a diplomate of the American Board of Orthopedic Surgery, fellow of the American College of Surgeons and of the New York Academy of Medicine; a member of the American and International Orthopedic Associations, the American Academy of Orthopedic Surgeons, the American Medical Association, the New York County and State Societies, a member and former president of the Alumni of Bellevue Hospital. His clubs include the University, the Tuxedo, and the Beaver Kill Trout Club.

In his "retirement" Dr. Krida has worked out a busy, pleasant and highly satisfactory way of living. He is in his office or visiting hospitals or operating five days a week. But Friday noon he leaves his office, not to return until Monday noon. These long week ends and six to eight weeks in the summer he spends at his country place at Tuxedo, New York, about 40 miles from New York City. Driving his own car, he covers the distance in about an hour and a quarter. The place is a remodelled carriage house on eight acres of what was once one of the great estates of the area. After buying it in 1944 Dr. Krida had a lot of fun playing architect—it now has a "Romeo and Juliet" balcony where the hay-lift used to be, two Dutch fireplaces (one in the living room and one in the upstairs bedroom) and a flagstone terrace.

At Tuxedo, Dr. Krida sweats at his hobby of amateur forestry and landscaping. He likes to grow trees, and thins them out scientifically; he wields ax and saw on those he decides should be cut down. He hauls firewood (oak preferred, especially white oak) back to New York for the woodburning fireplace in his Manhattan apartment, which is around the corner from his Park Avenue offices. He likes to raise flowers too, and has won prizes for dahlias at the Tuxedo Horticultural Shows, but admits that his gardener, Americo La Bruno, does the work—"all I do is look at them." The gardener works all year round and sees that the oilburner in the house is turned up in time for the Kridas' winter week-end visits. The country place is named

"Kribrouck," for Krida and the name of his second wife, the former Elsie Hasbrouck, whom he married in 1935. He has two sons, Arthur and Robert, by his first marriage to the former Marta Barkowska, whom he married in 1914; neither one is a doctor.

The former humus pit at the Tuxedo place, already equipped with water pipes for composting when Dr. Krida bought it, has been turned into a small swimming pool. But there is no pond big enough for game fish. Dr. Krida is enthusiastic when he discusses the orthopedic operations he has developed, but he really gets excited when he talks about fishing. His fishing, he says with a chuckle, has given him more fame among his colleagues than even his fascial band operation.

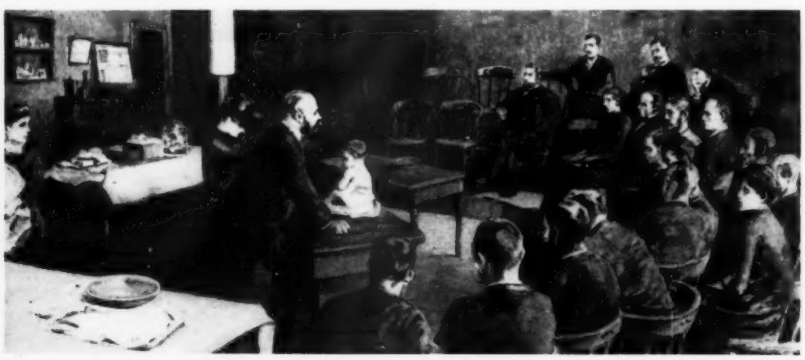
Dr. Krida usually goes to New Brunswick, Canada, every season, but he has missed the last two years, and has consoled himself by fishing for trout in the Catskills and for steelhead and bass in three Tuxedo lakes near his country home. He also likes photography, especially outdoor subjects, and prefers still photography to movies, which he tried and gave up. He likes to tell how, in New Brunswick, he once tossed rocks at a grazing bull moose to make it lift its head at just the right angle; he got the picture and escaped. This surprised the guide, who was calmly cooking lunch at the time, assuming that Dr. Krida was interested in fish, not moose. Among indoor activities Dr. Krida likes the theater and opera, and he is a regular Saturday afternoon listener to the Metropolitan's radio broadcasts.

While fishing on the Restigouche River in New Brunswick in 1943 he "killed" a 42 pound salmon, the third heaviest fish caught in "50 years of recorded annals" of the river. It was quite a battle. The salmon, 46 inches in length and 34 inches in girth, was caught on a No. 6 "Green Highlander" fly—a small fly, which added to the glory of eventual victory. On the first cast that day, the salmon missed the fly on its first try, and Dr. Krida gave himself and the salmon a rest by sitting in the canoe and smoking a cigaret. On the fourth cast the salmon, a female, finally took the fly, but he had to play it for 45 minutes before he could gaff and land it. (Unfortunately a bigger fish was hooked the next year, and Dr. Krida and his fish had to retire to fourth place.)

No advocate of ichthyologic orthopedics, Dr. Krida let the guide operate on his prize-winning salmon. The skin was pickled in a pail of brine after careful measurements had been taken for the taxidermist, who made a form for it like a shipbuilder's mold. Then the guide smoked the salmon, which was sent back to New York in an eminently edible condition. Mrs. Krida does not go on the Canadian fishing expeditions, but she enjoys eating the fish. So the Kridas were able to have their fish and eat it too; the salmon, mounted with the fatally enticing fly in its mouth, now hangs at the Tuxedo place alongside the portrait of the doctor painted by Irving S. Wiles.

It is a portrait of a successful doctor, who at 63 is briskly intent on doing the things he likes to do: to make the lame walk, and to persuade crippled trees to flower again. When not engaged in these things, he most likely can be found with a fishing rod in his hands, and a speculative glint in his eye—in another few weeks the trout will be running in the mountains, and there are even bigger salmon still uncaught.

George R. Burns



A case of spinal curvature. New York Polyclinic School of Medicine and Surgery. Drawing, 1887.